



جامعة صنعاء
كلية الطب والعلوم الصحية
دائرة العلوم السريرية الطبية
دفعة بصمه طبيب 31
طب بشري

OPHTHALMOLOGY



13 Strabismus
14 Optic nerve
19 Ocular manifestation
20 Intraocular tumors
21 Neuroophthalmology

part 4
Lectures

Lecture No.

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اللجنة العلمية Group C

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مندوب المجموعة

مسعد الصيادي

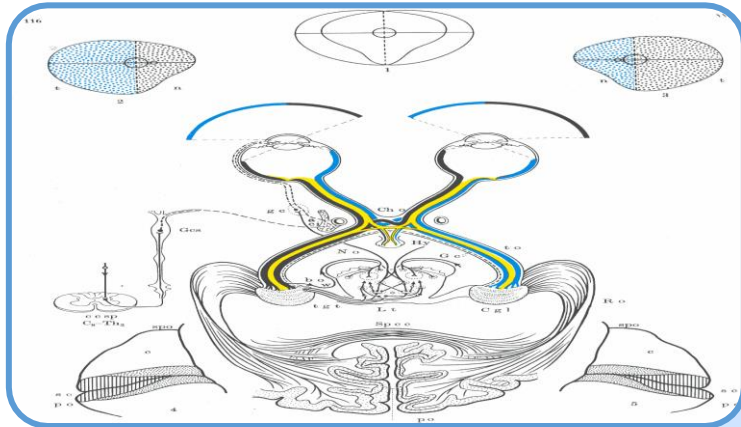
مع تحيات مكتبة الحزمي للخدمات الطلابية والمراجع الطبية
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VISIT...

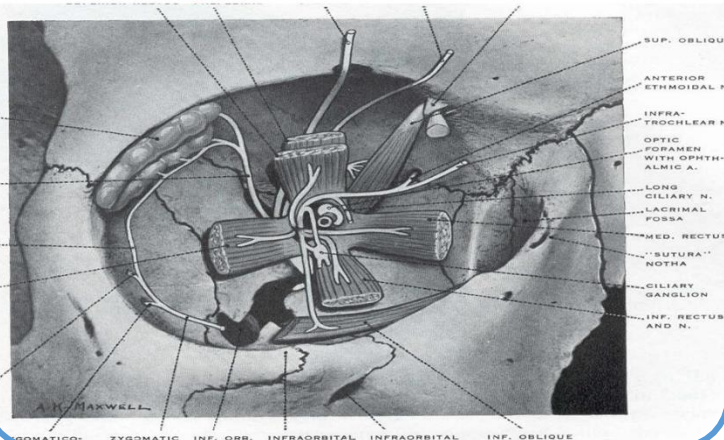
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Strabismus

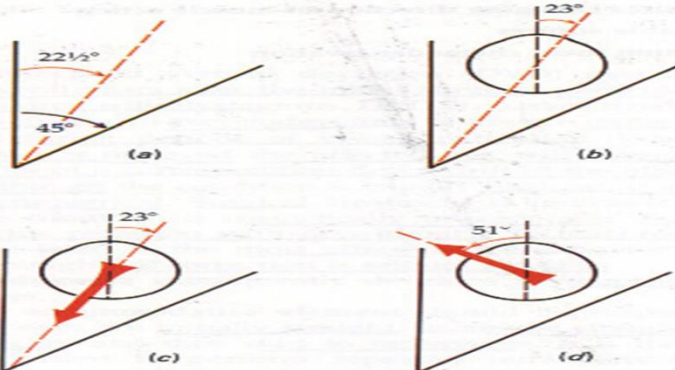
Abdulgohni O. Al-Barrag MD, Sana'a University, Sana'a



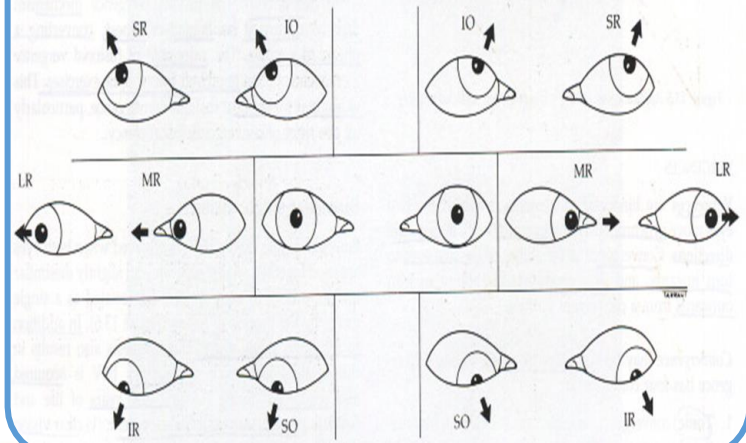
Anatomy of extra ocular muscles



Anatomy of extra ocular muscles



Action of extra ocular muscles



Classification of squint

- Apparent
- Real squint :
 1. Latent
 2. Manifest : Concomitant , Incomitant , & kinetic

Apparent squint

- Etiology :
 - Epicanthus
 - Abnormal IPD
 - High errors of refraction

Epicanthus

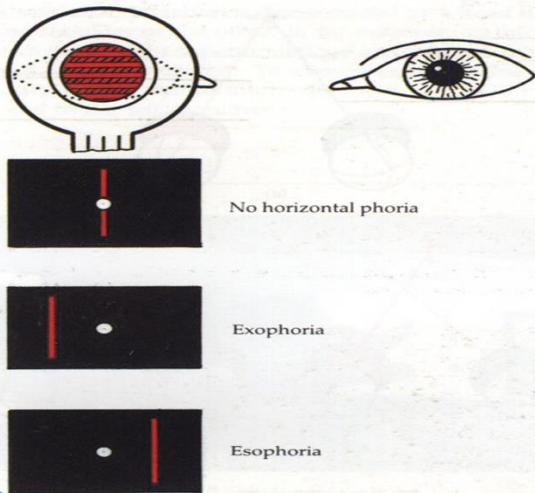


Heterophoria

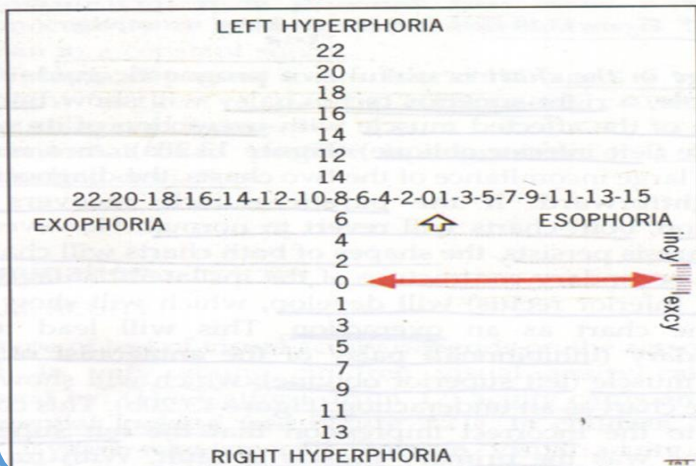
- Etiology : Errors of refraction, & weakness of one or more than one muscle
- Symptoms : Asthenopia , running letters , & diplopia.

- Diagnosis :Cover test ,Maddox rod & Maddox wing.
- Treatment :Glasses ,Prisms & surgery

Maddox rod



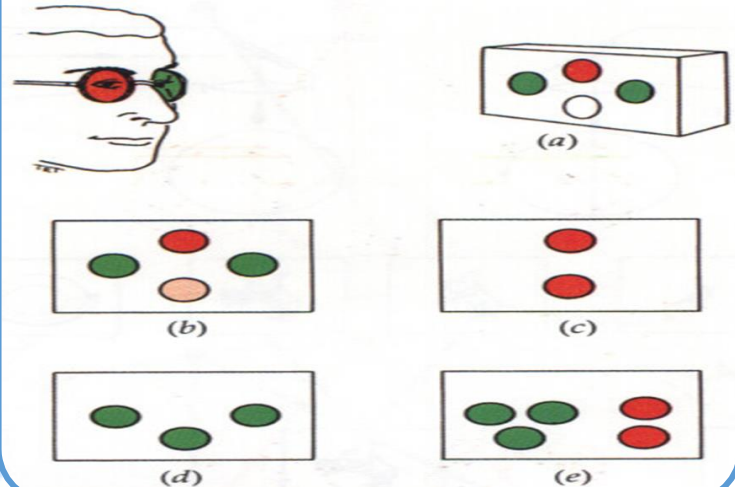
Maddox wing



Incomitant squint

- Etiology: Nuclear ,Nerve & muscle lesion
- Signs & symptoms:
 1. Squint & limitation of movements
 2. Diplopia
 3. False projection
 4. Compensated head posture
 5. Headache ,nausea ,vomiting & vertigo

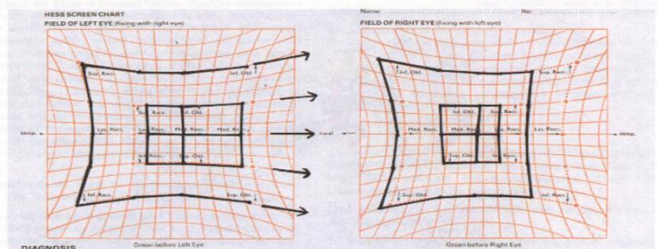
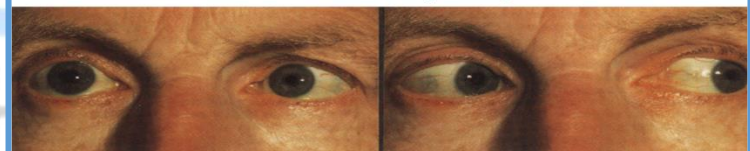
Worth's four-dot test



Incomitant squint

- Diagnosis :
 1. Ocular motility
 2. Diplopia chart
 3. Hess screen
- Treatment: Treatment of the cause ,& surgery

Abducens nerve palsy

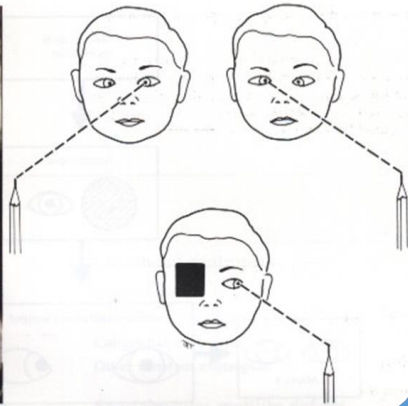


Concomitant squint

- Etiology : Theory
- Types : Unilateral ,alternating &periodic
- Sequelea: Suppression ,amblyopia &eccentric fixation

- Diagnosis :Visual acuity ,cover test ,angle of squint ,binocular vision & fundus examination

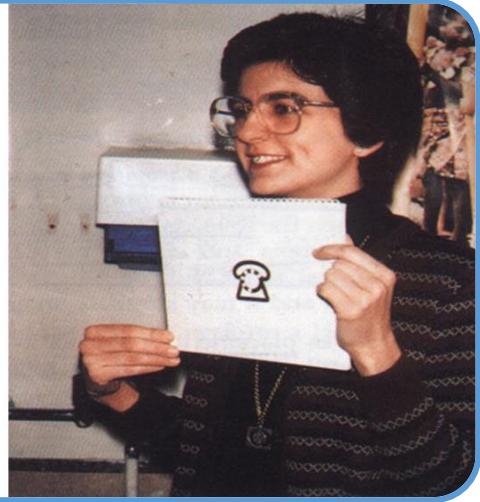
Esotropia



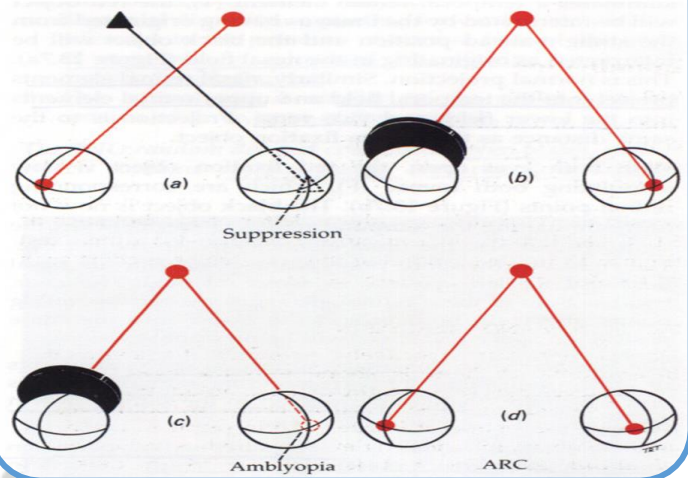
Right exotropia



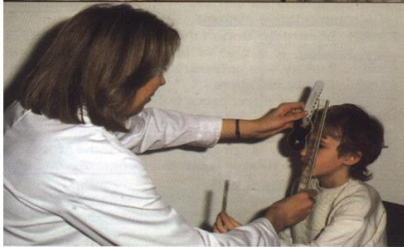
Visual acuity



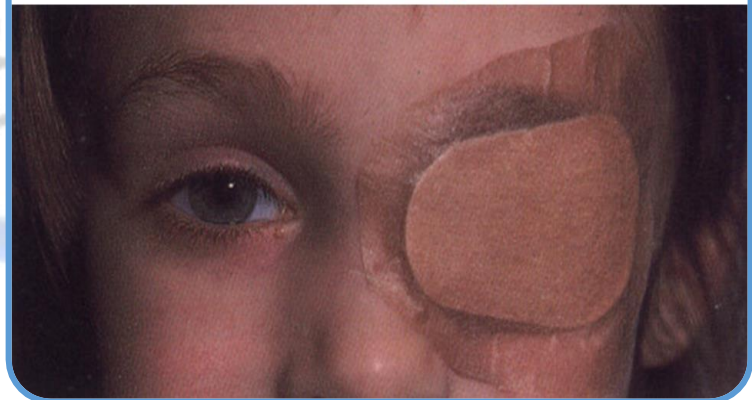
Amblyopia



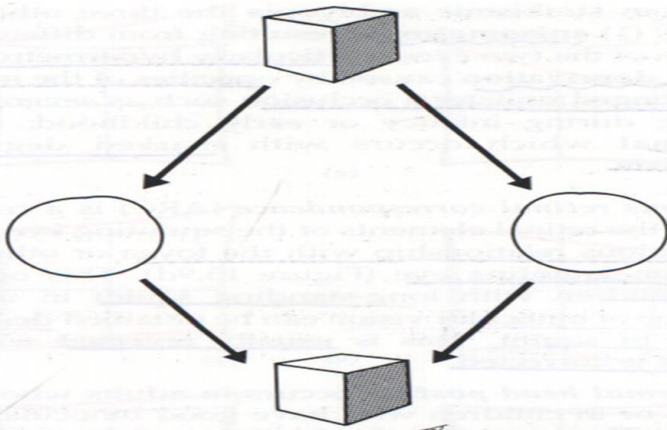
Cover test



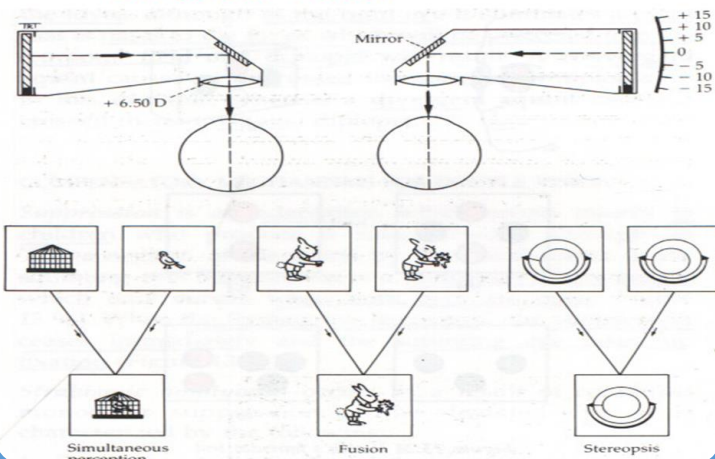
Treatment of Amblyopia



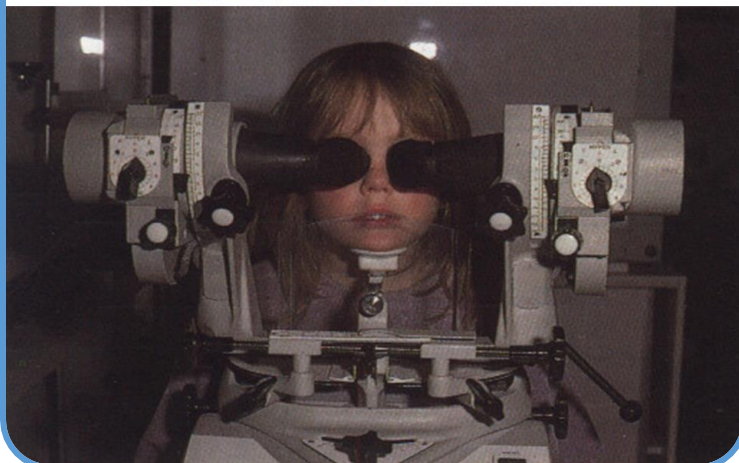
Binocular vision



Binocular vision



Binocular vision



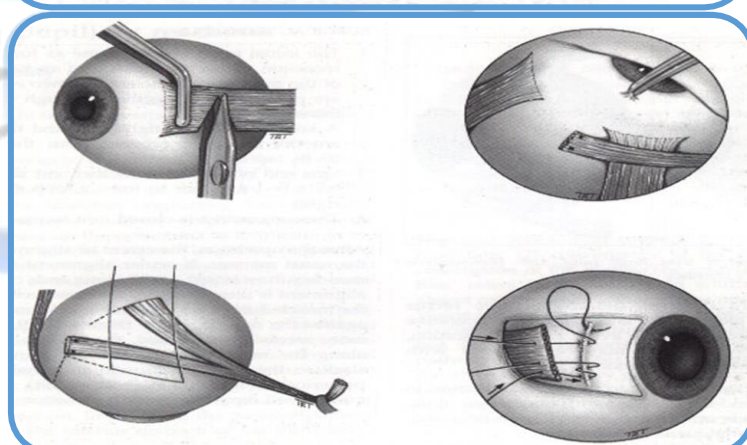
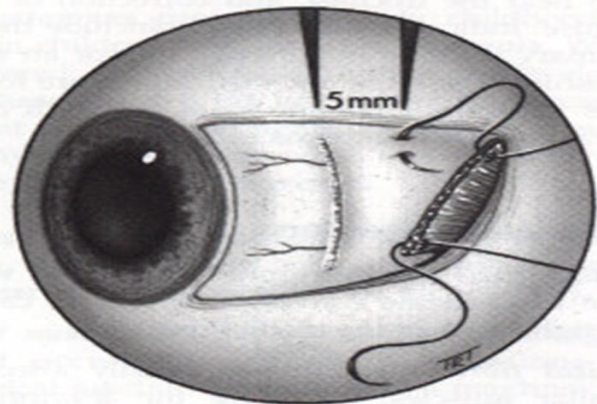
Kinetic squint

- Irrigative of the brain without true of paralysis (meningitis, encephalitis, epileptic fits & brain tumors)

Squint surgery

- Principles of strabismus surgery
- The aims of surgery on the extraocular muscles are to correct the misalignment of the eyes and, if possible, also to restore BSV. The three main types of operation are the following:
 - Weakening procedures which decrease the pull of a muscle.
 - Strengthening procedures which enhance the pull of a muscle.
 - Procedures which change the direction of the action of a muscle.

Recession of horizontal rectus muscle



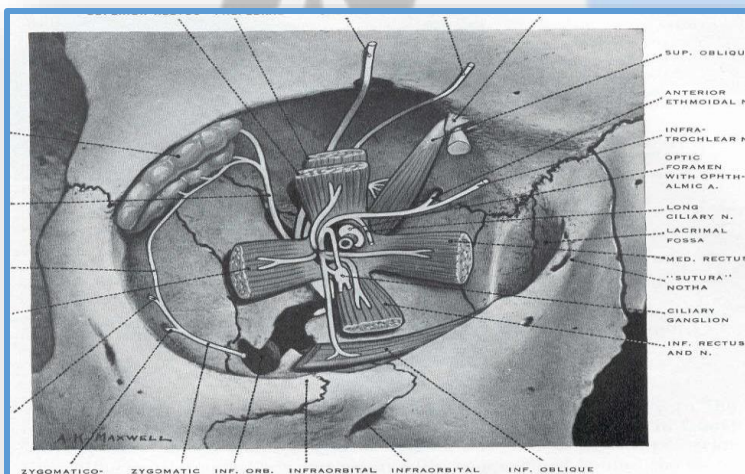
STRABISMUS

Mahfouth A Bamashmus FRCSEd FRCOphth
Sana'a University Medical School

Anatomy of Extraocular Muscles

- Six muscles
- 4 Recti: MR, LR, SR, IR
- 2 Obliques: SO, IO
- Origin:
 - Recti and SO: Common origin from the annulus of Zinn (tendinous ring at the apex of the orbit)
 - IO: Floor of orbit

Anatomy of Extraocular Muscles



Anatomy of Extraocular Muscles

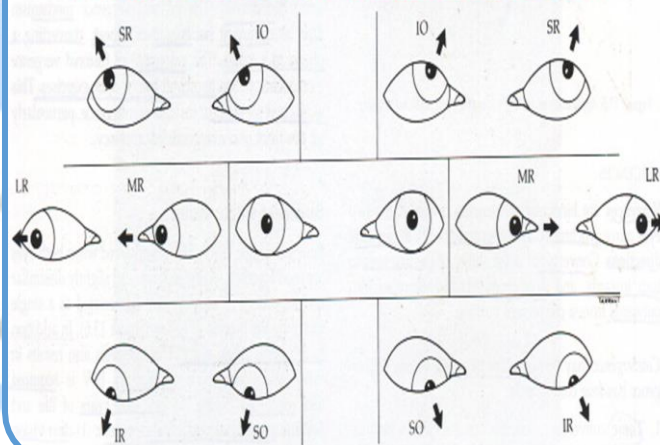
- Insertion (inserted in the sclera)
 - MR: 5.5 mm from limbus
 - IR: 6.6 mm from limbus
 - LR: 7.0 mm from limbus
 - SR: 7.7 mm from limbus
 - SO: upper posterior-lateral of the sclera
 - IO: lower posterior-lateral of the sclera
- Blood supply: muscular branches of ophthalmic artery
- Nerve supply
 - Oculomotor (3rd):
 - Superior division: SR
 - Inferior division: MR, IR, IO

- Trochlear (4th): SO
- Abducent (6th): LR

Actions

Actions	Main Action	Subsidiary Action
MR	Adduction	
LR	Abduction	
SR	Elevation	Adduction and Intorsion
IR	Depression	Adduction and Extorsion
SO	Depression	Abduction and Intorsion
IO	Elevation	Abduction and Extorsion

Action of extra ocular muscles



Binocular Movements

1. Conjugate movements

Movement of the two eyes in the same direction with the visual axes parallel
2. Vergence movements

Movement of the two eyes in opposite directions in which the visual axis do not remain parallel

Vergence Movements

- ❖ Convergence
 - Convergence of the 2 visual axes to a near object
- ❖ Divergence
 - Divergence of the 2 visual axes on looking to a far object
- ❖ Synergistic Muscles
 - Muscles which contract together
(RLR & LMR when eye moves to the R side)
- ❖ Antagonistic Muscles
 - Muscles which relax together
(RMR & LLR when eye moves to the R side)

Strabismus (Squint)

- True Squint
 - Ocular deviation with abnormal direction of the visual axes of both eyes relative to each other
- Apparent Squint (pseudo-squint)
 - Appearance of ocular deviation but the visual axes of both eyes are normally directed relative to each other

Apparent Squint (pseudo-squint)

- Appearance of ocular deviation but the visual axes of both eyes are normally directed relative to each other
- Aetiology:
 - ☐ Epicanthus
 - ☐ High Myopia
 - ☐ Narrow / wide interpupillary distance (IPD)
 - ☐ High Hypermetropia
 - ☐ Ptosis

Epicanthus



Pseudo-deviations

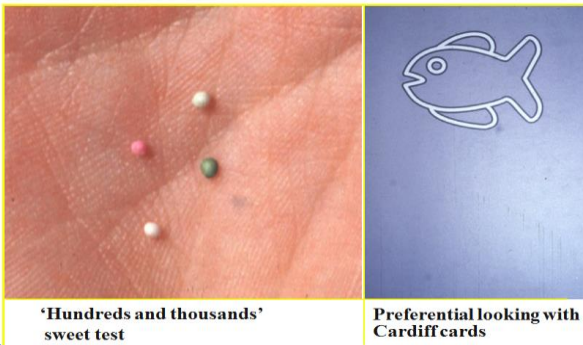
Pseudo-Esotropia	Pseudo-exotropia
 • Epicanthic folds • Short interpupillary distance • Negative angle kappa	 • Wide interpupillary distance • Positive angle kappa

Squint

Diagnosis:

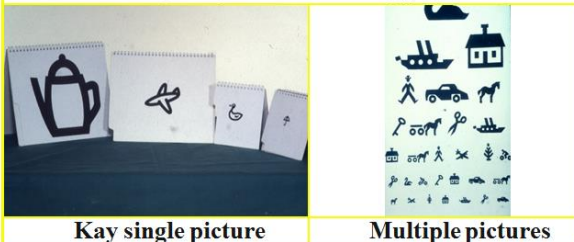
- Visual acuity
- Cover test
- Angle of squint
- Binocular vision
- Fundus examination

Visual acuity tests in preverbal children

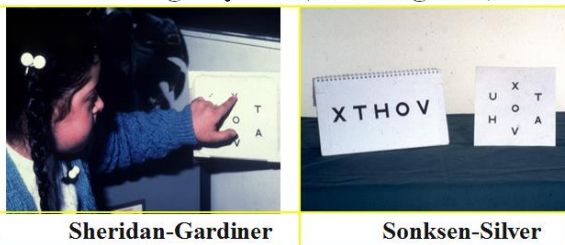


Visual acuity tests in verbal children

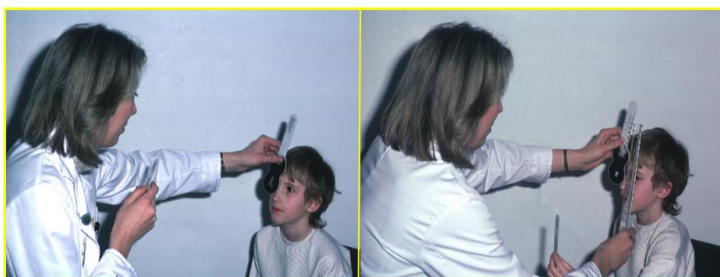
At age 2 years (naming pictures)



At age 3 years (matching tests)



Cover tests



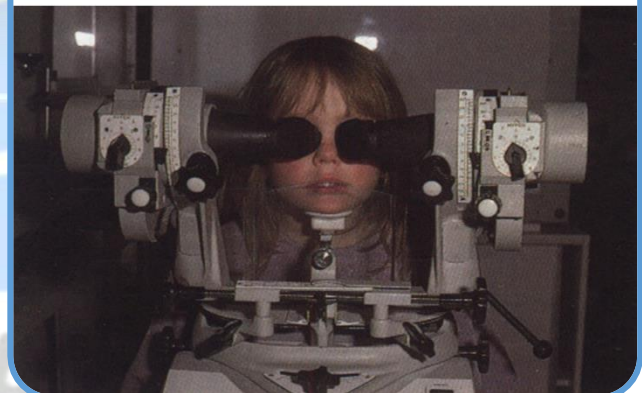
- **Prism cover test measures total deviation**

Hirschberg test

- **Rough measure of deviation**
- **Note location of corneal light reflex**
- **1 mm = 7 or 15**



Binocular vision



True Squint

1. Latent squint (heterophoria)
2. Manifest squint
 - a) Paralytic squint
 - b) Concomitant squint
 - c) Kinetic squint

Latent Squint

1. Exophoria: Tendency for eyes to deviate out
2. Esophoria: Tendency for eyes to deviate in
3. Hyperphoria: Tendency for eyes to deviate up
4. Hypophoria: Tendency for eyes to deviate down
5. Cyclophoria: Tendency for eyes to roll around

Latent Squint

Aetiology

- Errors of refraction
 - Myopia produces Exophoria
 - Hypermetropia produces Esophoria
- Weakness of one or more of the muscles

Symptoms

- Eye Strain
- Diplopia

Diagnosis:

- Cover Test
- Maddox Rod Test
- Maddox Wing

Dissimilar image tests

Maddox wing	Maddox rod
• Dissociates eyes for near fixation (1/3 m) • Measures heterophoria	• White spot converted into red streak • Cannot differentiate tropia from phoria

Latent Squint

Treatment

- Correct of error of refraction
- Exercising prisms
- Surgical Treatment

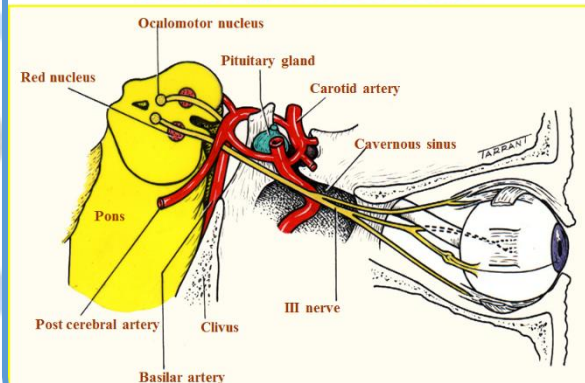
True Squint

- Latent squint (heterophoria)
- Manifest squint
 - Paralytic squint
 - Concomitant squint
 - Kinetic squint

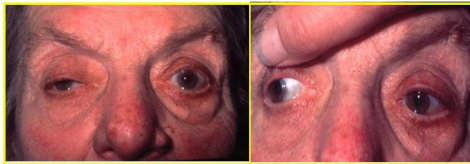
Paralytic Squint

- True manifest squint due to paralysis of one or more of the extraocular muscles and the angle of squint is VARIABLE in different directions of gaze (Incomitant Squint)
- Aetiology: 3rd, 4th and 6th nerve palsy
 - Nuclear lesions
 - Nerve lesions
 - Muscle lesions
- Symptoms
 - Diplopia
 - Ocular deviation with limitation of movements of the affected eye
 - Ocular torticollis (head rotation &/or tilt)
 - Vertigo, dizziness, uncertain gait, nausea, vomiting
- Signs
 - Ocular deviation and angles of deviation
 - Limitations of movements
 - Binocular diplopia
 - False projection (past pointing)
 - Ocular torticollis (compensatory head posture)
- Diagnosis
 - Examination of ocular motility
 - Hess screen

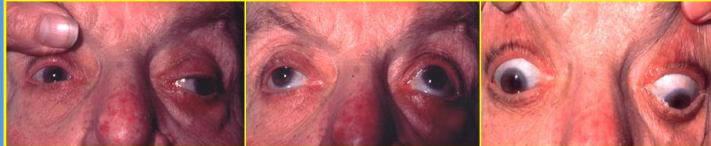
Anatomy of third nerve



Signs Of Right Third Nerve Palsy



- Ptosis, mydriasis and cycloplegia
- Abduction in primary position
- Normal abduction
- Intorsion on attempted downgaze

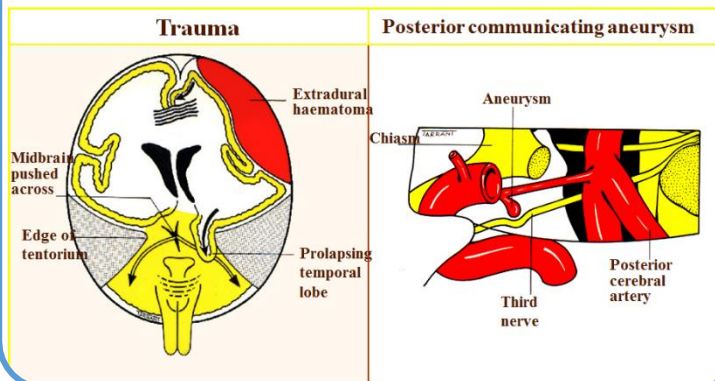


- Limited adduction
- Limited elevation
- Limited depression

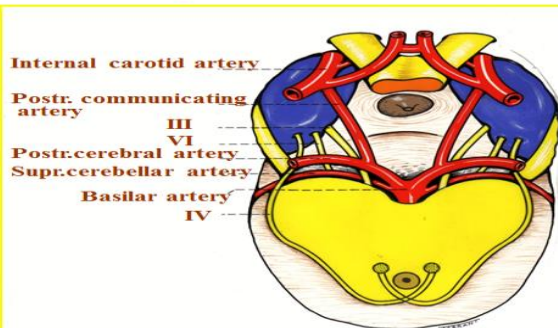
Important Causes Of Isolated Third Nerve Palsy

Idiopathic - about 25%

Vascular disease - hypertension, diabetes



Anatomy Of Fourth Nerve



- Only cranial nerve to emerge dorsally
- Crossed cranial nerve
- Very long and slender

- Very long and slender
- Crossed cranial nerve
- Only cranial nerve to emerge dorsally

Signs Of Right Fourth Nerve Palsy

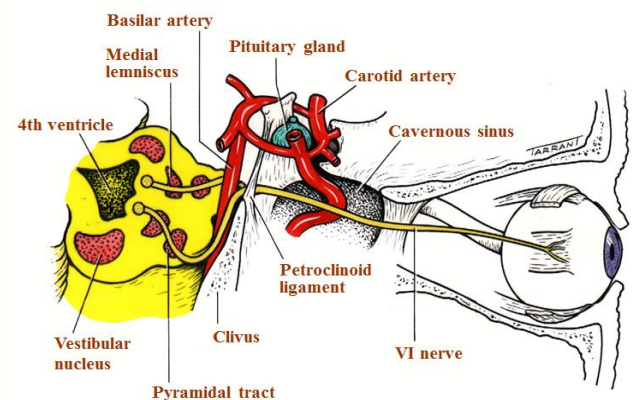


- Right hyperdeviation in primary position when left eye fixating
- Excyclotorsion
- Right underaction on depression in adduction
- Vertical diplopia



- Right overaction on left gaze

Anatomy Of Sixth Nerve



Recent Right Sixth Nerve Palsy



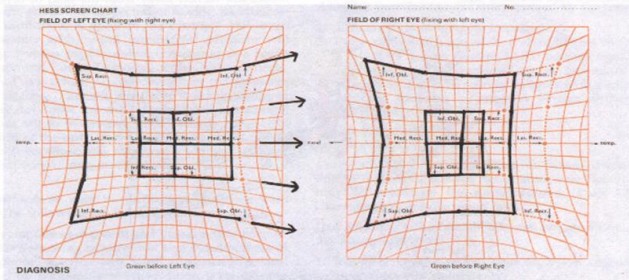
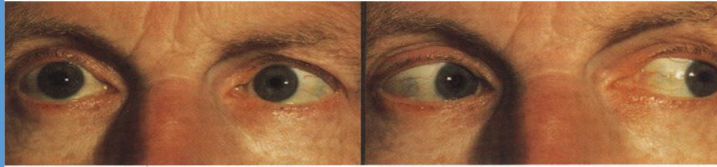
Right esotropia in primary position due to unopposed action of right medial rectus

Marked limitation of right abduction due to right lateral rectus weakness

medial rectus due to unopposed action of right medial rectus

Marked limitation of right abduction due to right lateral rectus weakness

Abducent (6th) Nerve Palsy



Concomitant Squint

Manifest ocular deviation with no muscle paralysis or limitation of movement present

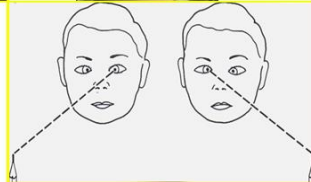
Essential Infantile Esotropia

Presents within first 6 months

Signs



- Angle large and stable
- Nystagmus in some cases
- Normal refraction for age
- Poor potential for BSV
- Amblyopia in about 30%



• Cross fixation

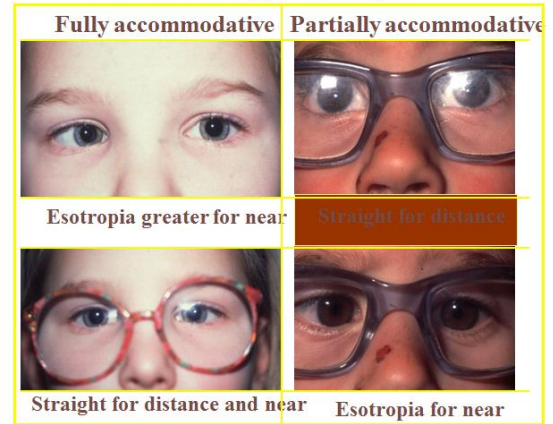
Management Of Essential Infantile Esotropia



- Correct amblyopia if present
- Surgery before age 12 months
- Bilateral medial rectus recessions
- Ideal alignment within 10

Refractive Accommodative Esotropia

- Presents between 18 months - 3 years
- Initially intermittent
- Normal AC/A ratio
- Excessive hypermetropia



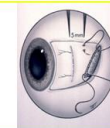
Management of accommodative esotropia

Refraction - prescribe full cycloplegic refraction under age 6 years

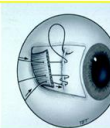
Treatment of amblyopia



Surgery - if spectacles do not fully correct deviation

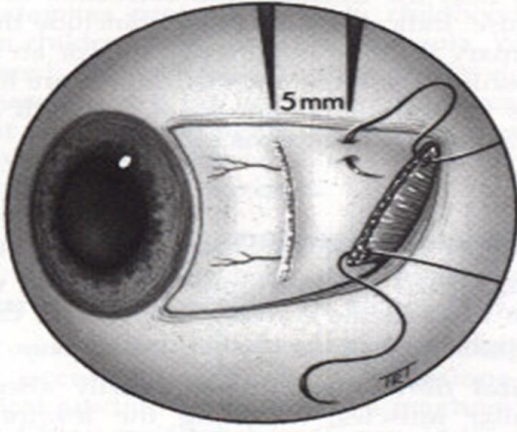


Recession



Resection

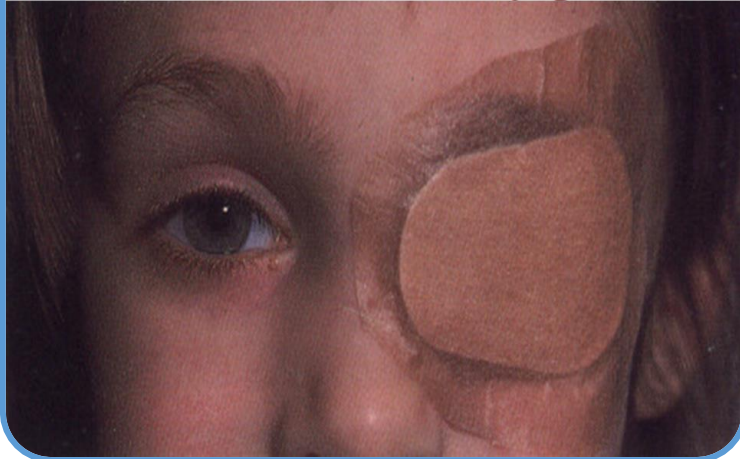
Recession Of Horizontal Rectus Muscle



Kinetic squint

- Irritative lesion of the brain without true paralysis (meningitis ,encephalitis ,epileptic fits & brain tumors)

Treatment Of Amblyopia



إضافات من محاضرة الدكتور عزيز شاهر

Binocular Vision

✚ **Coordinated:** use of 2 eyes > single image.

✚ **Development:**

- 0-6 months (fully developed fovea).
- 6 years (fully developed binocular vision).

✚ **Requirement:**

1. Clear ocular media (clear images).
2. Balanced muscles actions (images on fovea).
3. Well developed visual areas of the brain (fusion of images).

Binocular Vision Importance:

1. Stereopsis.
2. Larger visual field.
3. Field defect in one is masked by other overlapping field.

Latent Squint (Heterophoria)

Abnormal visual axes direction when binocular vision is dissociated

Paralytic squint

Abnormal visual axes direction > variable squint angle in different gaze direction

Paralytic Squint

Diagnosis

1. S&S
2. Ocular movement
3. Diplopia chart
4. Hess screen

Treatment

1. Treatment of the cause
2. Occlusion of one eye # diplopia
3. Surgical treatment

Concomitant Squint

Abnormal visual axes direction > constant squint angle in different gaze direction.

THANK YOU

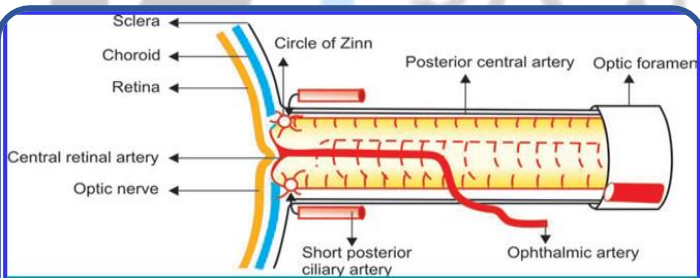
Optic Nerve

Applied Anatomy

- It extends from the lamina cribrosa upto the optic chiasma.
- The optic nerve originate from the nerve fibre layer of the retina. All the retinal fibres converge to form the optic nerve about 5 mm to the nasal side of the macula lutea. It pierces the lamina cribrosa to pass backwards and medially through the orbital cavity. It then passes through the optic foramen, backwards and medially to meet the nerve from the other eye at the optic chiasma.
- The optic nerve is covered with the meningeal sheaths after it pierces the lamina cribrosa.
- These meningeal spaces are continuous with those in the brain.
- The total length of the optic nerve is 5 cm. It can be divided into four parts:
 1. Intraocular — 1 mm
 2. Intraorbital — 25 mm
 3. Intracanalicular — 4-10 mm
 4. Intracranial — 10 mm

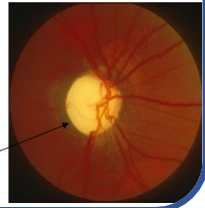
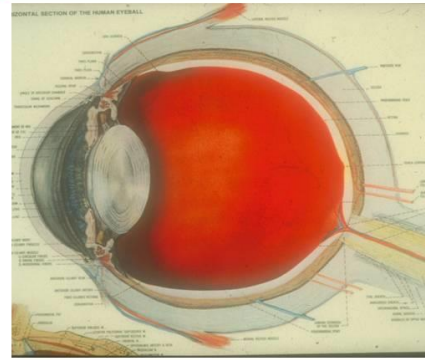
Optic Disc

- It represents the optic nerve head. It has only nerve fibre layer so it does not excite any visual response—"blind spot". It is a pink, oval or circular disc of 1.5 mm diameter. There is a depression in its central part which is known as the "physiological cup". It occupies the central one-third of the optic disc. Therefore, the normal cup: disc ratio is 1:3 or 0.3.



Blood supply of optic nerve

Normal Eye and Optic Disc



Cupped disc

Blood Supply

- The intraocular and intraorbital parts are supplied by the branches of the ophthalmic artery, short posterior ciliary arteries and central retinal artery forming circle of Zinn.
- The *intracanalicular and intracranial parts are supplied by the branches of the anterior cerebral artery and ophthalmic artery.*

Venous Drainage

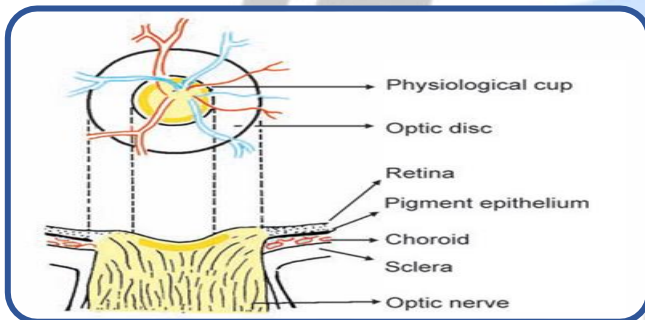
- It is by the central retinal vein and superior and inferior ophthalmic veins.

Functions

1. Optic nerve is responsible for normal visual acuity and field of vision.
2. It also acts as a filter and functions as,
 - i. Afferent pupillary pathway
 - ii. Colour vision and light brightness appreciation.

Diseases Of The Optic Nerve

1. Vascular disturbances—Papilloedema.
2. Inflammation
 - a. Acute
 - i. Papillitis (optic neuritis)
 - ii. Retrobulbar neuritis
 - iii. Neuroretinitis
 - b. Chronic—The toxic amblyopias.
3. Degeneration—Optic atrophy.
4. Tumours—Glioma, meningioma.
5. Congenital anomalies
 - a. Coloboma Fundus oculi examination
 - b. Medullated nerve fibres.



Papilloedema (Choked Disc)

It is a hydrostatic, non-inflammatory oedema of the optic disc. It results from increased intracranial pressure and venous stasis.

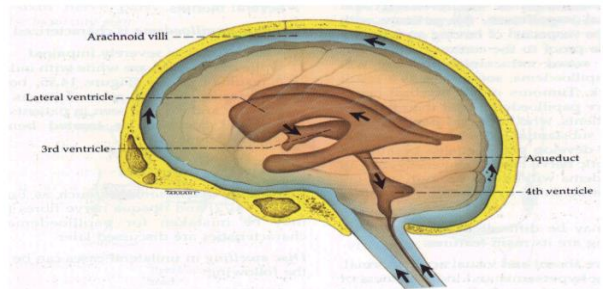
Etiology

1. Increased Intracranial Pressure

It is the most common cause of bilateral papilloedema. It may be associated with the following conditions:

- A. Intracranial space occupying lesions—These include space occupying lesions, as brain tumour, abscess, aneurysm,
 - Subdural haematoma hydrocephalus, etc. The tumour of cerebellum, midbrain and parietooccipital region produce papilloedema more rapidly than the lesions involving other area.
 - The fast progressing lesions produce papilloedema more frequently and acutely than the slow growing lesions.
 - **Foster-Kennedy syndrome**—The frontal lobe, pituitary and middle-ear tumours such as meningiomata of the olfactory groove are sometimes associated with,
 - i. Pressure atrophy of the optic nerve on the side of the lesion due to direct pressure.

Circulation of cerebrospinal fluid



ii. Papilloedema on the other side due to the effect of generalized raised intracranial pressure.

- B. Systemic conditions :include malignant hypertension, toxemia of pregnancy, cardiopulmonary insufficiency, blood dyscrasias and nephritis.
- C. Cerebral or subarachnoid haemorrhage can give rise to a papilloedema
- D. Meningitis
- E. Encephalitis, cerebral oedema and encephalopathies.
- F. Pseudotumour cerebri—It is an important cause of raised intracranial pressure. It is a poorly understood condition, usually found in young obese women. It is characterised by chronic headache and bilateral papilloedema without any localising neurological signs.

2. Orbit Lesions

The orbital space occupying lesions are frequently associated with papilloedema on the involved side such as tumours, orbital abscess and cellulitis, aneurysm of ophthalmic artery, pseudotumour and endocrinal exophthalmos.

3. Ocular Lesions

These include marked ocular hypotony, acutely raised intraocular pressure, central retinal vein occlusion, anterior ischaemic optic neuropathy and uveitis.

Pathogenesis

1. It is due to venous stasis which results in the compression of the central retinal vein as it crosses the subdural and subarachnoid spaces.
2. There is vasodilatation at the disc due to hypoxia causing axoplasmic stasis.
3. The nerve fibres at the optic disc become swollen which degenerate later on.

Unilateral versus Bilateral Papilloedema

- In majority of the cases with raised intracranial pressure, it is bilateral. Papilloedema due to ocular and orbital lesions is usually unilateral.
- However, unilateral cases as well as of unequal size do occur with raised intracranial pressure, e.g. Foster-Kennedy syndrome.

Symptoms

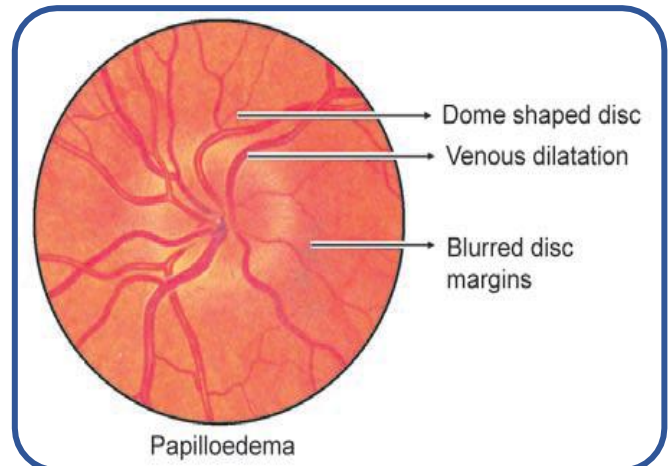
1. General symptoms include headache which is made worse by coughing, sneezing or straining.
2. Projectile vomiting (without nausea) is suggestive of raised intracranial pressure.
3. There are transient attacks of blurred vision (amaurosis fugax).
4. Central vision is affected only in late stages.

Signs

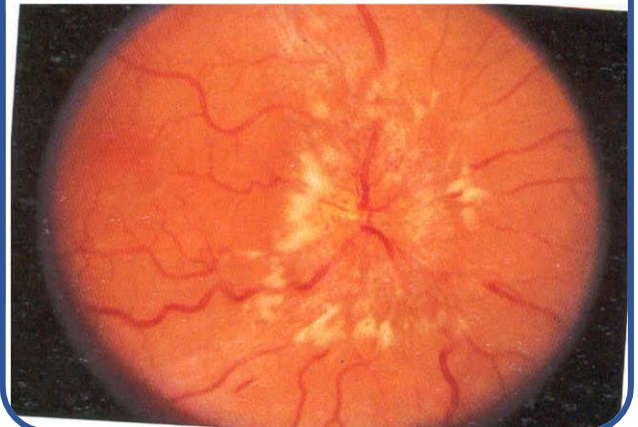
1. Fundus Examination

a. Early changes

- i. *Optic disc*—There is blurring of the disc margin.
 - o Physiological cup gets filled up.
 - o There is hyperemia of the disc with capillary dilatation.
 - o The disc becomes gradually elevated (mushroom or dome-shaped) so that the vessels bend sharply over its margin. There is a difference of 2-6 D between the vessels at the top and those on the retina. By indirect ophthalmoscopy, a definite parallax can be elicited.
 - o Edema gradually spreads to the surrounding retina.
- ii. *Vessels*—Veins are markedly congested, dilated and tortuous.
- iii. *Macula*—Macular star may be seen due to retinal oedema.
- iv. *General fundus*—Cotton wool soft exudates and both flame-shaped and punctate haemorrhages appear around the optic disc.



Papilloedema



Early Papilledma



- VA - normal
- Mild disc hyperaemia
- Indistinct disc margins - initially nasal
- Mild venous engorgement
- Normal optic cup
- Spontaneous venous pulsation - absent (also absent in 20% of normals)

Established Papilledma (Acute)



- VA - usually normal
- Severe disc elevation and hyperaemia
- Very indistinct disc margins
- Obscuration of small vessels on disc
- Marked venous engorgement
- Reduced or absent optic cup
- Haemorrhages + cotton-wool spots
- Macular star

Atrophic Papilledma (Secondary Optic Atrophy)



- VA - severely decreased
- Mild disc elevation
- Indistinct disc margins
- Disc pallor with few crossing vessels
- Absent optic cup

Signs

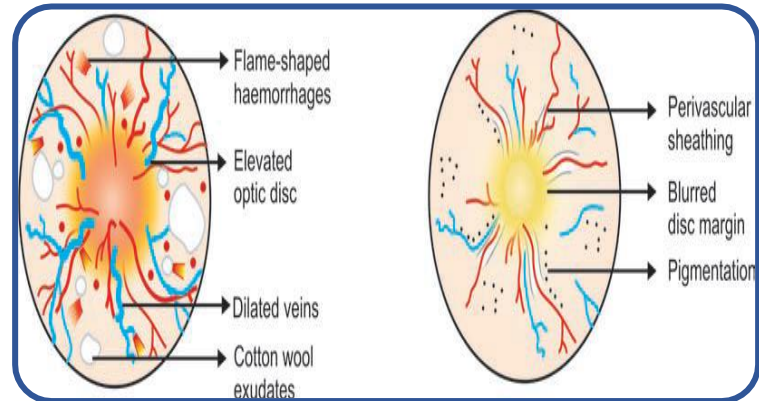
b. Late changes

- Postneuritic optic atrophy—The disc becomes pale with blurred margin.
- Thickening of the perivascular sheaths lead to contraction of arteries.
- Generalized retinal pigmentation may be present.

2. Visual Fields

- There is enlargement of the blind spot.
- There is progressive contraction of the visual fields.
- Complete blindness sets in eventually.

- Associated neurological symptoms include headache, nausea and vomiting in cases of raised intracranial pressure.



Differential Diagnosis

Blurring of the optic disc margin is seen commonly in cases of:

- Optic neuritis.
- Pseudoneuritis—in hypermetropia the lamina cribrosa is small and the nerve fibers are heaped up.
- Astigmatism.
- Malignant hypertension.
- Toxemia of pregnancy.
- Central retinal vein occlusion. Papilloedema and oedematous retina
- Drusen of the nerve head—It is a typically bilateral and inherited condition.

Treatment

- Treat the underlying cause of papilloedema.
- Surgical decompression should be done early, i.e. before the visual field changes occur.
- There is rapid recovery of vision after the decompression usually.

Optic Neuritis

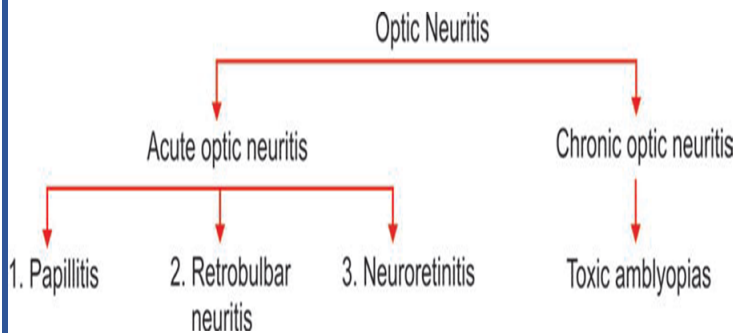
The inflammation of the optic nerve is known as optic neuritis.

Classification

It can be divided into two main types:

1. Acute optic neuritis
2. Chronic optic neuritis

OPTIC NEURITIS



Papillitis

Papillitis is an acute inflammation of the optic nerve head (papilla or optic disc) associated with rapid loss of vision. It is the ophthalmoscopically visible or anterior part of the optic nerve. It is often unilateral.

Etiology

A. Multiple sclerosis is the most common cause.

It causes demyelination of the optic nerve.

B. Other central nervous system diseases

1. Neuromyelitis optica of Devic
2. Acute disseminated encephalomyelitis
3. Herpes zoster
4. Epidemic encephalitis
5. Poliomyelitis
6. Leber's disease

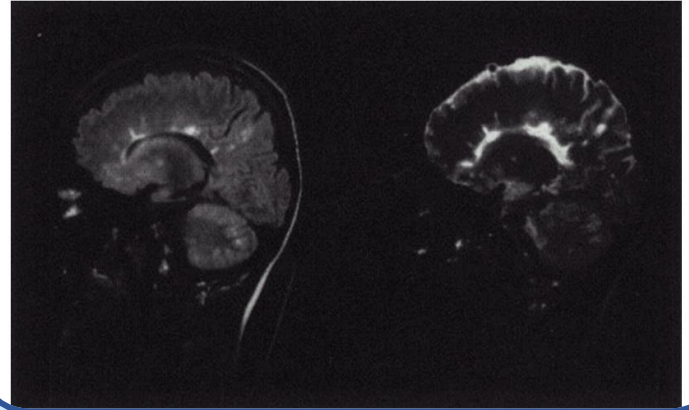
C. Local causes

1. Retinitis
2. Uveitis
3. Meningitis

D. Endogenous causes

1. Acute infections such as influenza, (measles, mumps, etc.)
2. Septic foci in teeth, tonsils, throat, etc.
3. Metabolic conditions, e.g. as in diabetes, anaemia, starvation.

Sagittal MRI scan showing periventricular plaques of demyelination. Left: T1-weighted; right: T2-weighted



Common Causes Of Sudden Loss Of Vision

1. Optic neuritis—Papillitis, retrobulbar neuritis
2. Central retinal artery occlusion
3. Central retinal vein occlusion
4. Retinal detachment
5. Acute congestive glaucoma
6. Vitreous haemorrhage
7. Anterior ischaemic optic neuropathy (AION)
8. Cortical lesions—Trauma, vascular
9. Acute corneal hydrops as in keratoconus
10. Photo-ophthalmia—Eclipse and snow blindness, exposure to bright arc or flash light

Pathogenesis

There are inflammatory changes in the nerve (true optic neuritis) or in the sheath (perineuritis).

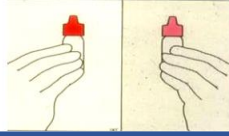
There is infiltration and loss of myelin sheath. Finally, degenerative changes and reactionary gliosis occur.

Symptoms.

1. Transient blurring of vision may be present initially.
2. Profound visual loss is the most important clinical feature
3. There is usually unilateral involvement.
4. There is sudden onset and rapid progress of the disease process.
5. Complete blindness sets in rapidly in untreated cases.

Clinical Examination

- Visual Acuity
- Colour Vision
- Visual Fields
- Pupils



Signs

1. **Pupil**—Direct light reflex is sluggish or absent as the afferent path is involved.

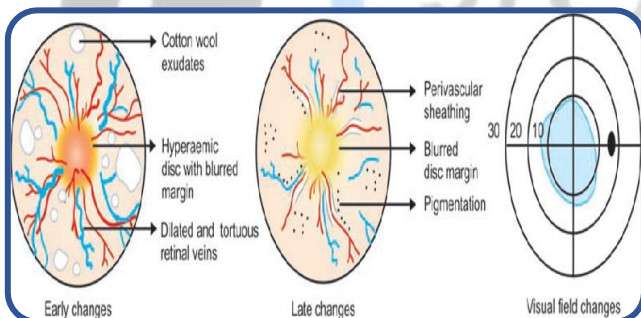
- Indirect light reflex and near reflex are present.

2. **Fundus Examination**

a. **Early changes**

These are similar to papilloedema. However, the disc swelling is usually upto 2-3D.

- Optic disc is hyperaemic with blurred margin.
- The swelling spreads to the surrounding retina.
- Retinal veins are tortuous and distorted.
- Exudates are present at the disc and retina.
- Vitreous is cloudy with fine opacities.
- Neuroretinitis—When the serious inflammation spreads from the disc towards the neighbouring retina, it is called neuroretinitis.



b. **Late changes**

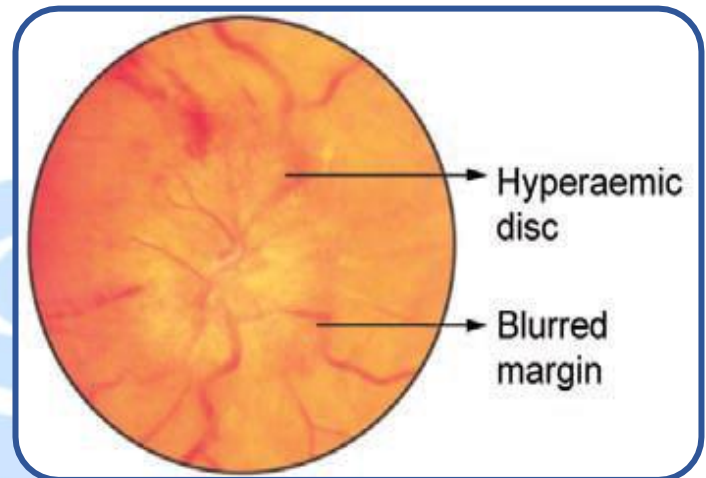
- Postneuritic atrophy sets in which is similar to papilloedema.
- Disc margin is blurred.

iii. Physiological cup gets filled up with organized fibrous tissue.

iv. Perivascular sheathing is usually present.

3. **Visual Field Defects**—A generalized depression of the visual field is the most common of visual defect.

Central, centrocaecal or paracentral scotoma may be present.

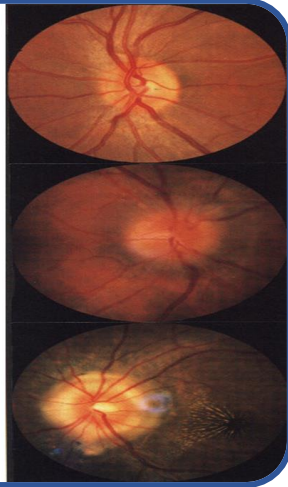


Treatment

- Efforts should be made to find out and treat the underlying cause of papillitis.
- There is no effective treatment for idiopathic and hereditary optic neuritis and that associated with demyelinating disorders.
- Corticosteroids may help in early recovery
 - Oral prednisolone therapy alone is contraindicated in the treatment of acute optic neuritis, since it was not shown to improve visual outcome and recurrence rate is high with this regime.
 - A patient presenting with acute optic neuritis should have MRI scan of the brain. If the brain shows lesions supportive of multiple sclerosis, the patient should receive immediate intravenous methylprednisolone (1 gm daily) for 3 days followed by oral prednisolone (1 mg/kg/day) for 11 days.
 - Indications for intravenous methylprednisolone in acute optic neuritis patients with a normal brain MRI scan are:
 - Bilateral simultaneous optic neuritis
 - Involvement of only good eye.
- Vitamin B1, B6 and B12 injections are given in full doses. Hydroxycobalamine (B12) acts as a detoxicating agent.

Classification of Optic neuritis

1. Retrobulbar neuritis - normal disc.
2. Papillitis.
3. Neuroretinitis.



Retrobulbar Neuritis

It is an acute inflammation of the optic nerve situated behind the eyeball.

Symptoms

These are same as for papillitis.

Sudden, profound loss of vision is the most common presenting complaint.

Common Causes Of Sudden Painful Loss Of Vision

1. Acute Congestive Glaucoma
2. Retrobulbar Neuritis
3. Temporal Arteritis

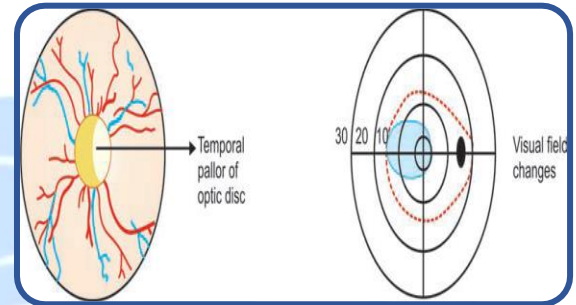
Signs

1. Fundus examination
 - "Neither the ophthalmologist nor the patient sees anything".
 - No ophthalmoscopically visible changes are seen.
 - If the lesion is situated near the lamina cribrosa, there is slight distension of the veins and attenuation of the arteries.
 - Temporal pallor of disc (due to involvement of papillomacular bundle) may be present occasionally.
2. Local pain on moving the eye is often present.
3. Marcus Gunn pupil—there is lack of sustained constriction of the pupil to light in swinging flashlight test. It indicates an afferent pupillary defect. It is a diagnostic sign. Swinging flashlight test—A bright light is thrown on to one pupil and its constriction is noted. The light is rapidly transferred to the other pupil after 2-3 seconds. This process of swinging of light to and

fro across the pupils is repeated several times so that there are equal impulses sent to the midbrain via the optic nerves.

4. Field of vision—Central, paracentral, sectorial scotomas or ring-shaped scotoma around fixation point may be present
5. Early loss of colour vision and contrast sensitivity may be present due to involvement of optic nerve.

Retrobulbar Neuritis



Complications

- 4- Recurrences are common specially in demyelinating diseases.
- 5- Complete blindness may occur eventually.

Differential Diagnosis

1. Malingering—It is seen in persons who hope to gain some advantage by pretending to be visually defective or handicapped. When one eye is said to be blind and there is absence of objective signs
2. Hysteria—History of psychiatric illness is very important.
3. Anterior ischaemic optic neuropathy (AION)—There is sudden loss of vision due to ischaemia of anterior part of optic nerve.

Treatment

It is same as for papillitis.

1. Retrobulbar injection of dexamethasone is very effective.
2. Systemic corticosteroids are given in full doses.
3. Vasodilators (systemic or local) may be effective.
4. Vitamin B1, B6 and B12 administered in high doses are useful adjunct.

Toxic Amblyopias (Chronic Retrobulbar Neuritis)

These include a number of conditions in which optic nerve fibres are damaged by the exogenous poisons such as:

1. *Mild toxic agents*—Tobacco, ethyl alcohol, carbon disulphide, iodoform, etc. They produce central scotoma due to initial effect on papillomacular bundle.
2. *Severe amblyopia*—It is produced by quinine organic arsenic, etc. They produce marked peripheral contraction of the visual field or even blindness.

TOXIC AMBLYOPIAS (CHRONIC RETROBULBAR NEURITIS)

It is frequently bilateral and has a chronic course with permanent visual deterioration. These can be divided broadly into two groups mild and severe. The following toxic agents can be involved:

6. Tobacco
7. Ethyl alcohol
8. Methyl alcohol
9. Lead
10. Quinine
11. Chloroquine
12. Ethambutol
13. Oral contraceptives.

1- TOBACCO AMBLYOPIA

✚ Etiology

It is caused by excessive use of tobacco by smoking and chewing. The toxic agent is the 'cyanide' found in tobacco leaves.

✚ Incidence

1. Age—35-50 years age group is prone to get this disease.
2. It is a bilateral condition usually.

✚ Predisposing Factors

1. General debility and malnutrition.
2. Digestive disturbances lead to malabsorption of vitamins.
3. Deficiency of vitamin B12 in dietary sources.
4. Associated alcoholism leads to deficient intake of vitamins and liver disorders.

✚ Pathogenesis

There is degeneration of the ganglion cells of the retina specially in the macular region.

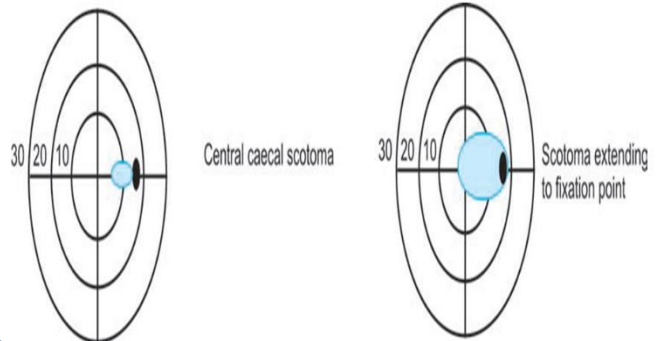
✚ Symptoms

1. There is increasing foginess of the vision.
2. Central vision is impaired so that there is difficulty in reading and doing near work.

✚ Signs

1. Fundus examination—It is normal or it may show slight temporal pallor of the disc.
2. Field of vision—There are characteristic field defects in the central field.
 - i. Central-caecal scotoma is present between the fixation point and the blind spot.
 - ii. It gradually extends to the fixation point and the central vision is lost.

Visual Field Defects In Tobacco Amblyopia



✚ Treatment

1. Complete withdrawal from tobacco and alcohol is a must.
2. General nutrition is improved.
3. Injections of vitamin B1, B6 and B12 are given in high doses. B12 injection 1000 mg are given intramuscularly biweekly × 3 weeks.

✚ Prognosis

It is good if it is treated early and adequately.

2- ETHYL ALCOHOL

It usually occurs along with the tobacco amblyopia. However, it may occur alone.

There is avitaminosis due to malnourishment and hepatic disorders.

Alcoholic peripheral neuritis is often associated with ocular lesion.

Symptoms, signs and treatment are same as for tobacco amblyopia.

3- METHYL ALCOHOL

✚ Etiology

- It is caused by the intake of wood alcohol or methylated spirit.

- The toxic agent is the *formaldehyde found in methyl alcohol*.

Incidence

1. It usually occurs during prohibition.
2. It involves several persons at a time consuming the wood alcohol from the same source.

Pathology

There is degeneration of the ganglion cells of the retina.

Symptoms

1. *Acute form*—It causes nausea, headache, giddiness, coma and blindness.
2. *Chronic form*—The symptoms of acute form relapse with progressive loss of vision.

Signs

Fundus examination

1. The disc margin is blurred.
2. The blood vessels are markedly reduced in size.
3. Primary optic atrophy sets in the final stage.

Complications

Blindness and death occur due to acidosis occur eventually.

Treatment

1. *Immediate gastric lavage is given to wash away the methyl alcohol.*
2. *Administration of alkali—Soda bicarbonate is given by 5% intravenous drip or orally as there is acidosis.*
3. *Ethyl alcohol is given in small frequent doses, i.e. 90 cc every 3 hours × 3 days. It prevents acidosis and ocular symptoms.*

Mode of action

1. Ethyl alcohol prevents oxidation of methanol to formaldehyde.
2. It competes with methanol for the same enzyme, i.e. alcohol dehydrogenase.

4- QUININE

It differs from tobacco amblyopia as it may cause total blindness with small dose of even 60 mg of quinine in susceptible cases.

Signs

1. The pupils are dilated and fixed.
2. Deafness and tinnitus may be associated.
3. Fundus examination shows pale and atrophic disc with contracted retinal vessels and oedema.
4. Visual fields are contracted causing tubular vision.

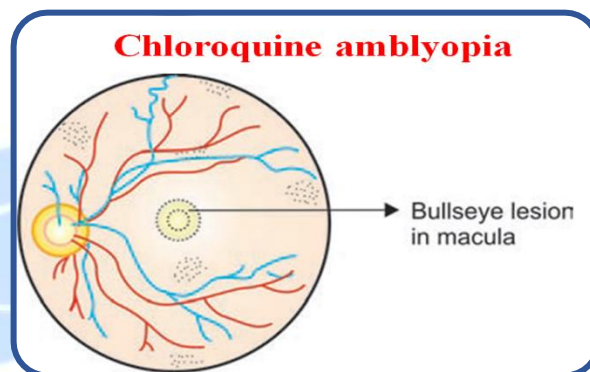
5- CHLOROQUINE

This is an antimalarial drug used in the treatment of lupus erythematosus and arthritis. The prolonged use of

chloroquine may cause keratopathy, myopathy and retinopathy.

Fundus examination

1. A mild pigmentary disturbance leads to the characteristic “bullseye” lesion in the macular area.
2. There is widespread retinal atrophy with clumps of pigment and attenuated retinal vessels seen in the later stage.



6- ETHAMBUTOL

Ethambutol is used in the treatment of tuberculosis. It may produce optic neuritis. The neuritis is reversible when the drug is stopped. The upper limit of safety dose is 15 mg/kg.

7- ORAL CONTRACEPTIVES

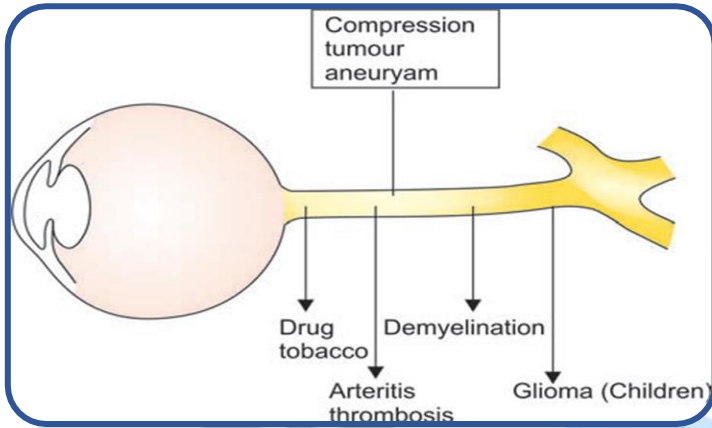
These are combination of progesterone and oestrogen. There is increased risk of vascular occlusion particularly in women who are suffering from hypertension, migraine or other vascular diseases.

There may be infraction of optic nerve head.

OPTIC ATROPHY

It is an atrophic condition of the optic disc whereby the optic nerve is degenerated. A diagnosis of optic atrophy depends on:

- Pallor of the optic disc
- Loss of visual acuity
- Defect in visual field



Pathogenesis

There is destruction of nerve fibres along with overgrowth of glial connective tissue.

1. Primary (Simple) Optic Atrophy

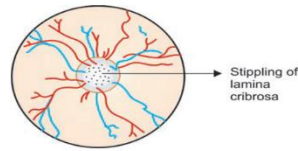
The lesion is proximal to the disc so there are no signs of local inflammation. It is associated with general disease usually of central nervous system

Etiology

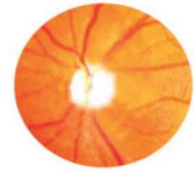
- Multiple sclerosis is a common cause.
- Tabes dorsalis due to syphilis is rare in recent times.

Fundus examination

- Optic disc is greyish-white or white in colour with clear margin. There is shallow, saucer-shaped atrophic cupping due to degeneration of nerve fibres. The stippling of lamina cribrosa may be seen.
- Retinal vessels and retina are normal.

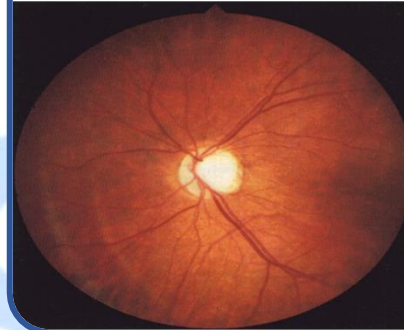


Primary optic atrophy



Primary optic atrophy

Primary Optic atrophy



- Multiple Sclerosis
- Toxic Amblyopia
- Trauma to Optic Nerve
- Syphilis

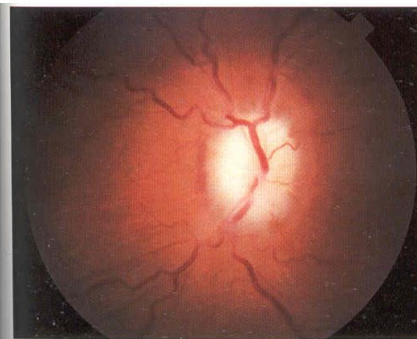
2. Secondary Optic Atrophy

Etiology

It follows any injury or direct pressure to the optic nerve from lamina cribrosa to the lateral geniculate body.

Fundus examination—It is same as for primary optic atrophy.

Secondary Optic atrophy



From previous swelling of optic nerve

- Post-Papilloedema
- Post-neuritis

3. Consecutive Optic Atrophy

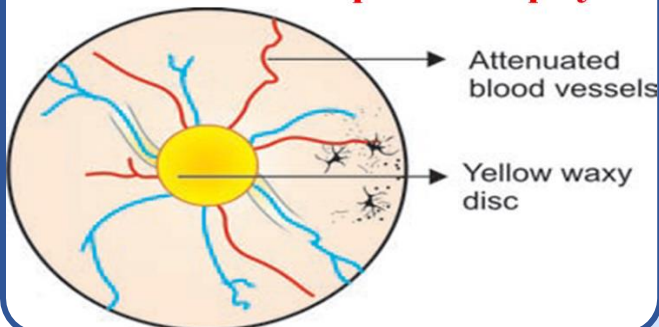
Etiology

Extensive retinal diseases cause ganglion cell destruction as occurs in retinitis pigmentosa and occlusion of central retinal artery.

Fundus examination

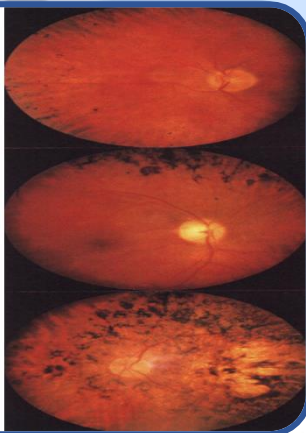
1. Disc is yellowish waxy in colour with less sharply defined margin.
2. Vessels are very much attenuated.

Consecutive Optic Atrophy



Consecutive Optic Atrophy

- ☐ Retinitis pigmentosa (RP)
- ☐ CRAO
- ☐ Myopia



4. Postneuritic Optic Atrophy

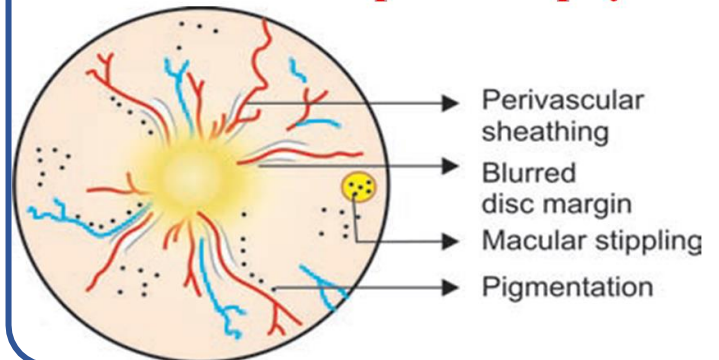
Etiology

It follows papilloedema and papillitis.

Fundus examination

1. Disc is pale along with blurred margin. The physiological cup is full.
2. Vascular sheathing is usually present.
3. Macular stippling may be seen.

Postneuritic Optic Atrophy



5. Ischaemic Optic Atrophy

Etiology

It is due to the central retinal artery occlusion.

Fundus examination—Disc and macular area are pale. Retinal vessels are obliterated.

6. Toxic Optic Atrophy

It has been already discussed under toxic amblyopias.

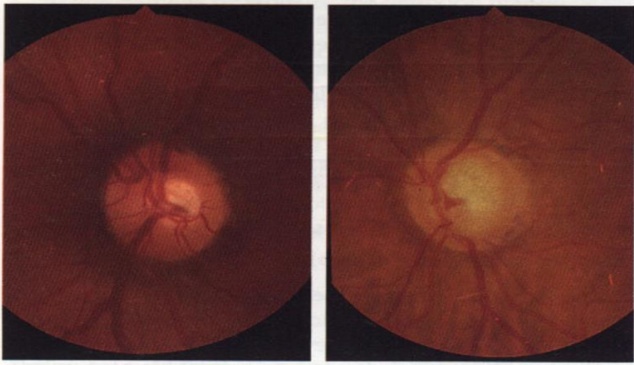
7. Glaucomatous Optic Atrophy

It has been already discussed under glaucomatous optic disc changes

Symptoms

1. In partial optic atrophy, the vision is markedly impaired.
2. In complete or total optic atrophy, the person is blind with no perception of light..

Glaucomatous Optic Atrophy



Signs

Pupil is dilated and fixed, i.e. not reacting to light.
Visual field shows concentric contraction with depression of central vision in initial stages with or without scotomata.

Treatment

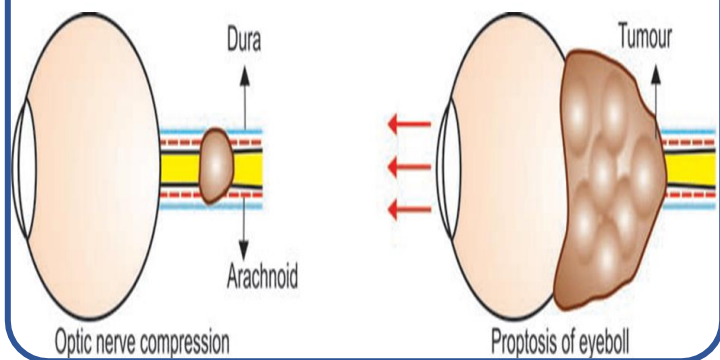
- Treat the underlying cause in cases of partial optic atrophy.
- No treatment is effective once complete optic atrophy has set in.

TUMOURS

Glioma

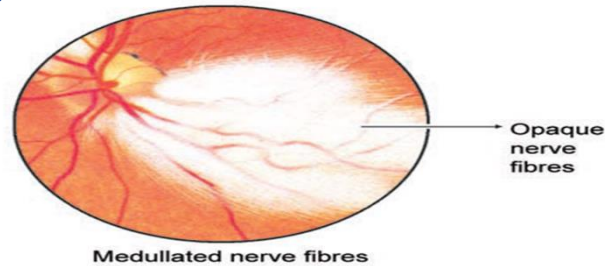
It is a congenital tumour occurring in the age group of 2-5 years. It is slow growing and self-limiting with good prognosis.

Tumours



Meningioma

It occurs in middle-aged women usually. There is early visual loss and proptosis. The prognosis is good because of the slow growth and peripheral situation of the tumour.



They appear as white glistening patches with radially striated edges. They are continuous with the optic disc.

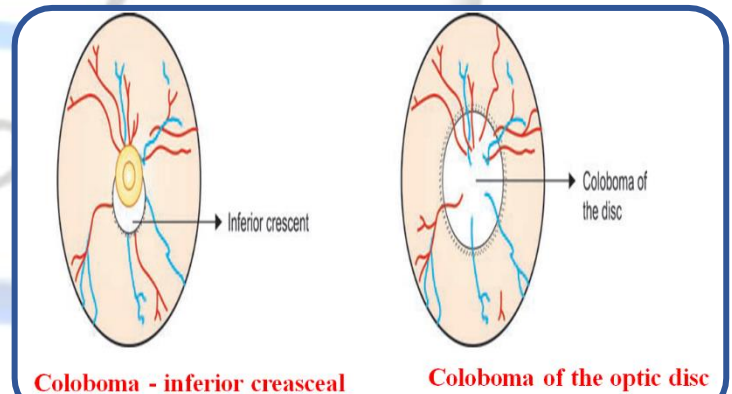
Congenital Anomalies

1. Coloboma of the Optic Disc

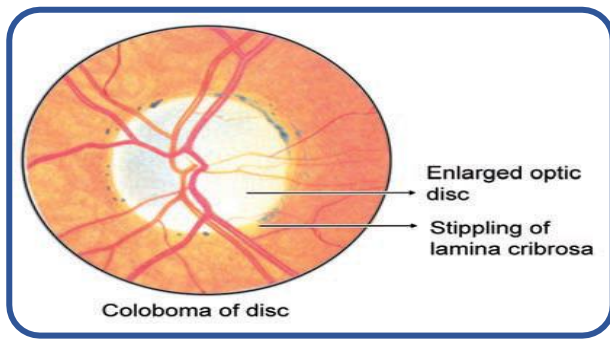
Colobomas are bilateral in more than half the cases. It occurs in two forms:

i. Inferior crescent

- This is a common form occurring due to incomplete closure of the embryonic fissure.
- It occurs in hypermetropic and astigmatic eyes.
- It may be ectatic.



- ii. *Coloboma of the disc*—There is a greater failure of the embryonic fissure to close. The apparent large disc is really the sclera. The vision is defective usually.

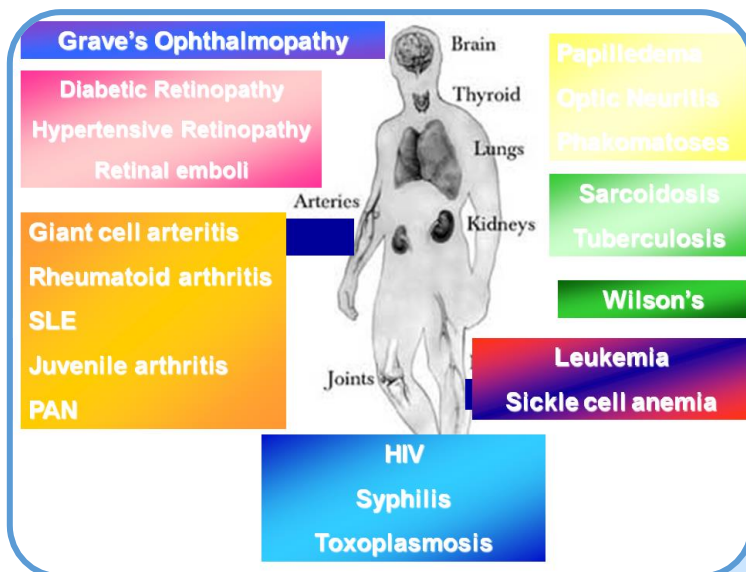


2. Medullated (Opaque) Nerve Fibres

Normally the myelin sheaths of optic nerve stop at the lamina cribrosa. Occasionally patches of nerve fibres regain these sheaths after they have passed through the lamina cribrosa.

Ocular Manifestations of Systemic Diseases

Dr. SALEH AL-AKILY Sana'a University
9-03-2019



Common Systemic Diseases Affecting The Eye

A. Infectious

1. Toxoplasmosis
2. Toxocariasis
3. TB
4. Syphilis
5. Leprosy
6. HIV
7. CMV

B. Non-infectious

1. Endocrine – diabetes, thyroid
2. Connective tissue disease – RA/ SLE/ Wegeners/ PAN/ Systemic sclerosis
3. Vasculitides (GCA)
4. Sarcoidosis
5. Behcet's Disease
6. Vogt Koyanagi Harada syndrome
7. Phakomatoses

RETINAL VASCULAR DISEASES

Diabetes And The Eye

EPIDEMIOLOGY OF D.M

Commonest cause of blindness in the population of working age in developed countries

Prevalence of DR of any severity in the diabetic population is 30% and prevalence of blindness due to DR is approximately 5%

Risk Factors

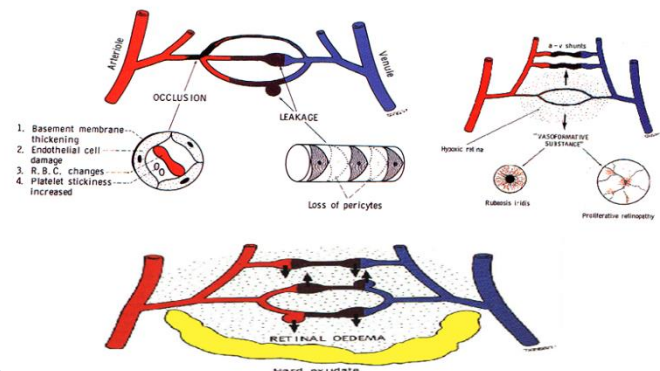
1. Duration of DM
2. Control of DM. Will not prevent but delays
3. Hypertension
4. Renal Disease
5. Pregnancy
6. Obesity, hyperlipidaemia, smoking, anaemia

Diabetic Retinopathy

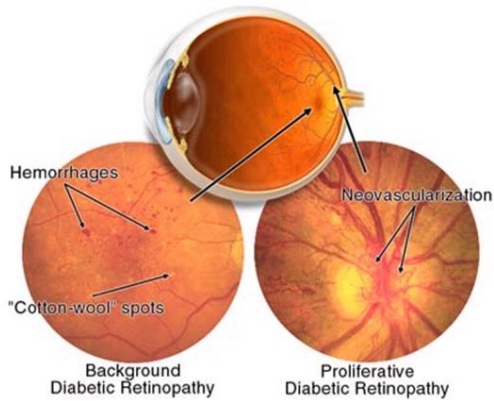
Pathogenesis :

- I. Microvascular occlusion
- II. Microvascular leakage

PATHOGENESIS



Diabetic Retinopathy



A More Recent Classification Of DR

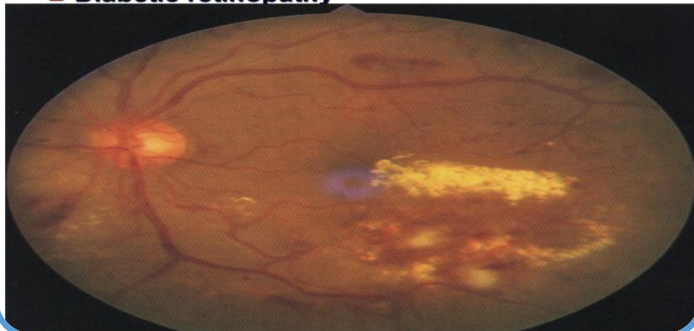
1. Nonproliferative DR (NPDR).
2. Proliferative DR (PDR).

Pre-proliferative



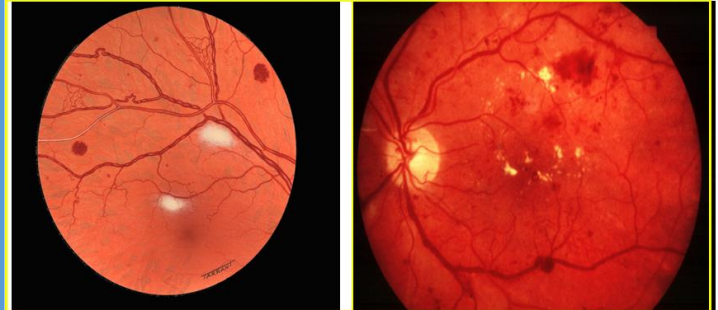
Endocrine diseases (Diabetes mellitus)

■ Diabetic retinopathy



Preproliferative diabetic retinopathy

Signs



- Cotton-wool spots
- Venous irregularities

- Dark blot haemorrhages
- Intraretinal microvascular abnormalities (IRMA)

Treatment not required but watch for proliferative disease

Proliferative Diabetic Retinopathy (PDR)

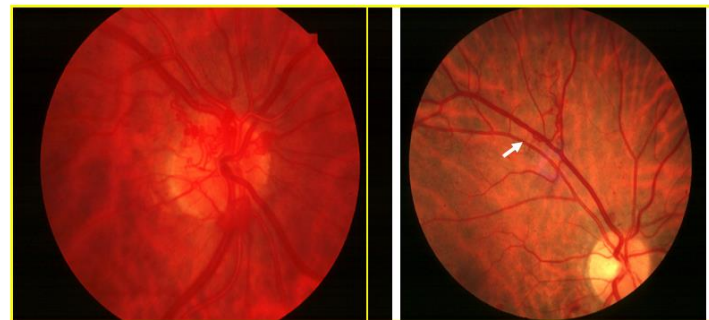
PDR = Neovascularization

Proliferative diabetic retinopathy

- Affects 5-10% of diabetics
- IDD at increased risk (60% after 30 years)

Neovascularization

- Flat or elevated
- Severity determined by comparing with area of disc

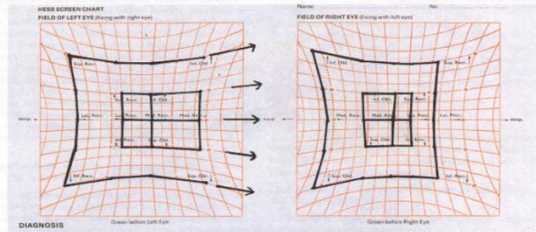
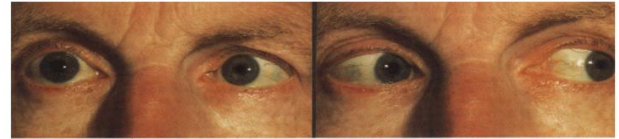


3) Diabetic retinopathy, proliferative



Massive neovascularization along the retinal surface (this indicates that the vitreous is not detached)

Endocrine Diseases (Diabetes Mellitus) Paralytic Squint



Endocrine diseases (Diabetes mellitus)

Rubeosis Iridis

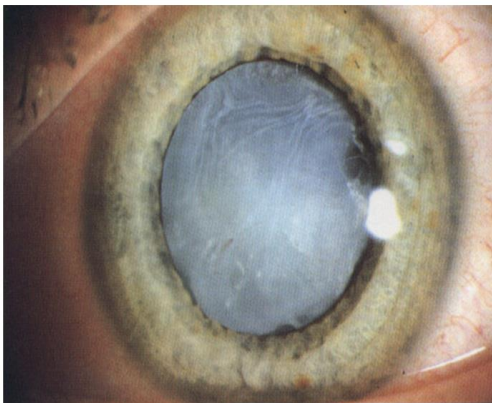


Other Retinal Vascular Disorders

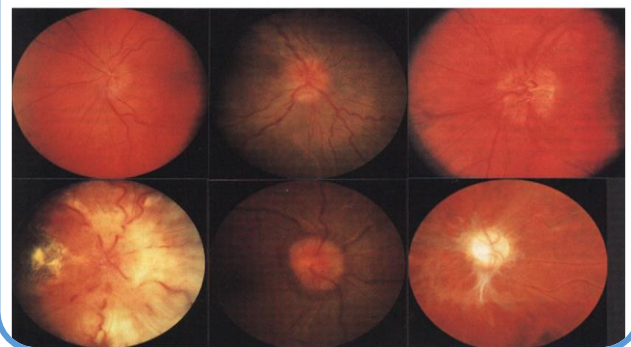
1. Retinal vein occlusion
 - a. Branch
 - b. Central
2. Retinal artery occlusion
 - a. Branch
 - b. Cilioretinal
 - c. Central
3. Hypertensive retinopathy
4. Sickle-cell retinopathy
5. Retinopathy of prematurity
6. Retinal telangiectasias
 - a. Idiopathic juxtafoveal
 - b. Leber miliary aneurysms
 - c. Coats disease
7. Retinal artery macroaneurysm
8. Radiation retinopathy

Endocrine Diseases (Diabetes Mellitus)

Pre-senile Cataract



Hypertension



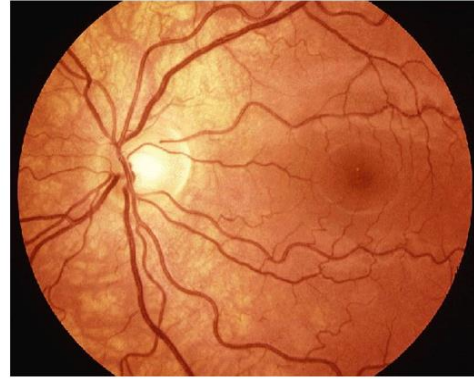
Hypertensive Retinopathy Classifications

Grade	Description
1	Mild retinal vascular changes (generalized arteriolar narrowing).
2	Moderate to severe retinal vascular changes (arteriovenous crossing and nicking changes).
3	Stage 1 and 2 findings, plus cotton-wool spots, retinal hemorrhages and exudates.
4	Stage 3 findings, plus associated optic nerve head swelling and macular star formation.

Prof M Bamashmus

Hypertensive retinopathy

Arteriovenous crossing changes.



Ocular Associations Of Hypertension



Retinal vein occlusion



Retinal artery macroaneurysm

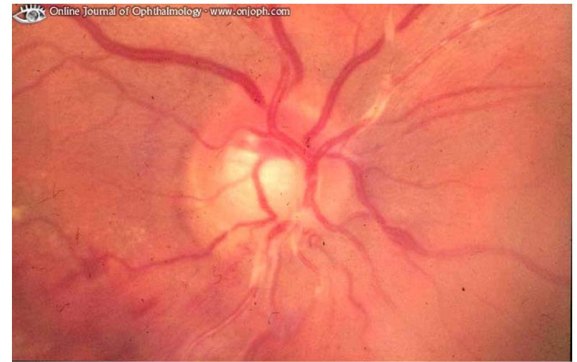


Anterior ischaemic optic neuropathy



Ocular motor nerve palsies

Arteriosclerosis of Retinal Vessels



Ensheathed arterial vessels are seen near the disc. Other signs of hypertension are small intraretinal hemorrhages, some exudates, slight edema of the inferior disc margin, and congested veins.

Hypertensive Retinopathy

Arteriolar constriction



Focal

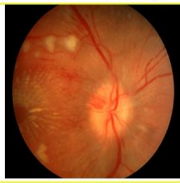
Generalized

Arteriolosclerosis (A-V changes)

Extravascular signs



Flame-shaped retinal haemorrhages

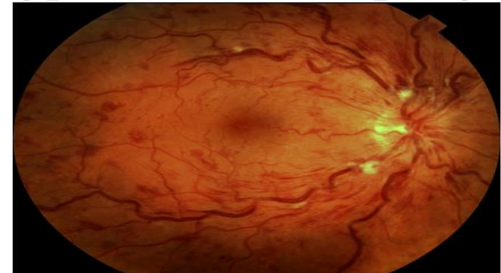


Cotton-wool spots and macular star



Disc oedema

Hypertensive retinopathy

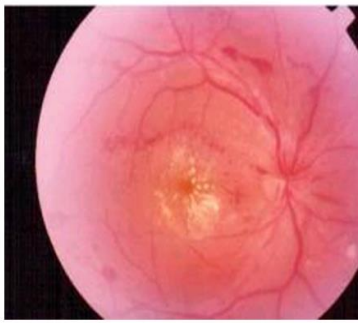


Hemorrhages ,Cotton wool patches

Hemorrhages ,Cotton wool patches

Hypertensive retinopathy

*Disc edema.
Macular star.*

**Severe Hypertensive Retinopathy****Hypertensive Retinopathy.**

Hemorrhages.

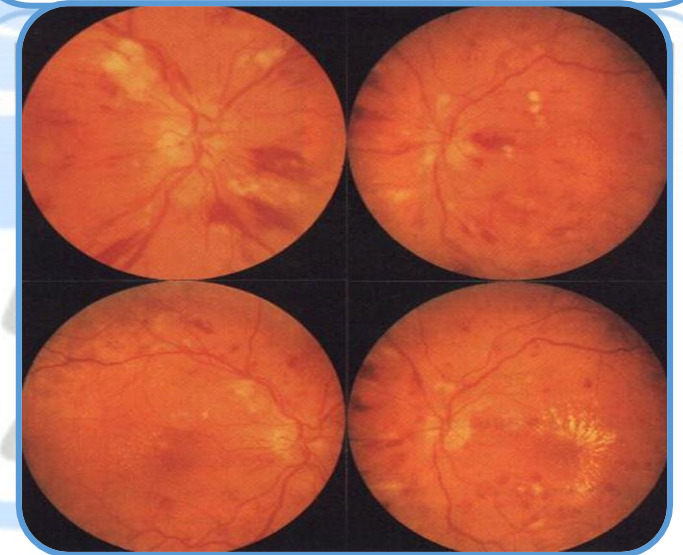


Macular star.

1) Hypertensive Retinopathy with Pre- and Intraretinal Hemorrhage

Centrally there is a well-defined accumulation of blood under detached internal limiting lamina. A second ring of blood has less well-defined borders and represents preretinal blood where the vitreous is detached from the retina.

Vitreous is detached from the retina.
Well-defined borders and represents preretinal blood where the detached internal limiting lamina. A second ring of blood has less
Centrally there is a well-defined accumulation of blood under

**Malignant Hypertension.**

Cardiovascular Diseases

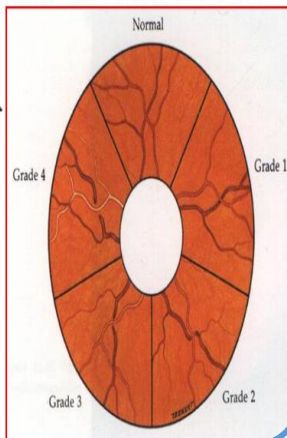
■ Retinal Arteriosclerosis Copper-wire arteries



Vascular Sclerosis Of The Retina

■ Arteriosclerosis changes :

Change in the vascular reflex .
Change in the course and diameter
Sheathing of the blood vessels
A/V. crossing changes



Cardiovascular Diseases

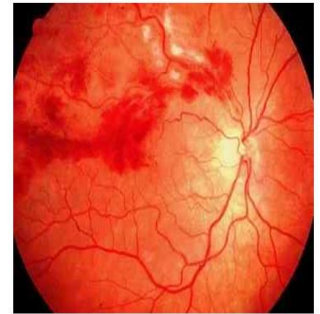
Retinal Arteriosclerosis Copper-wire arteries



Pathophysiology Of Retinal Venous Occlusions

Venous Occlusion → Stagnation → Hypoxia →
Edema & Hge. → ↑ Extravascular Pressure → More
Stagnation & So On

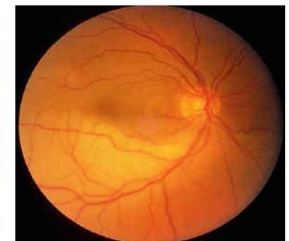
Retinal Vein Occlusion



Second most common cause of vascular-related visual loss.

Risk factors: hypertension, age, blood dyscrasias (OCP, HRT) and vasculitis (Behcets, sarcoidosis, AIDS, SLE)

Retinal Artery Occlusion

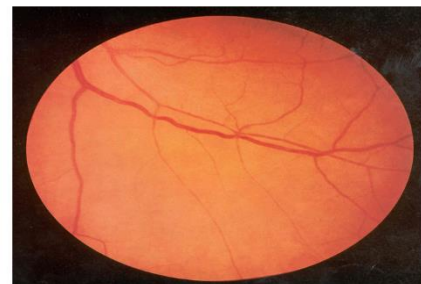


Risk factors: Carotid artery atherosclerosis (CRAO), carotid emboli (BRAO), vasculitis (GCA, SLE, PAN), coagulopathy.

OCULAR EMERGENCY - Immediate referral to ophthalmologist

Toxemia of pregnancy

1. Thread-like Arteries and Arterioles (due to vascular spasm)
2. Diffuse retinal edema



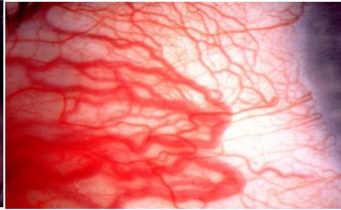
Thyroid Eye Disease

1. Soft tissue involvement
 - Periorbital and lid swelling
 - Conjunctival hyperaemia
 - Chemosis
 - Superior limbic keratoconjunctivitis
2. Eyelid retraction
3. Proptosis
4. Optic neuropathy
5. Restrictive myopathy

Soft Tissue Involvement

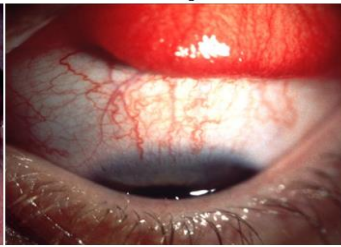
Periorbital and lid swelling

Conjunctival hyperaemia



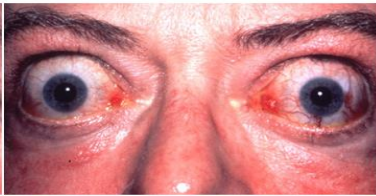
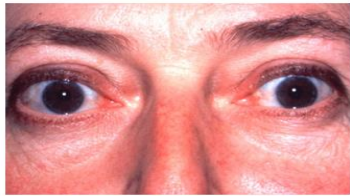
Chemosis

Superior limbic keratoconjunctivitis

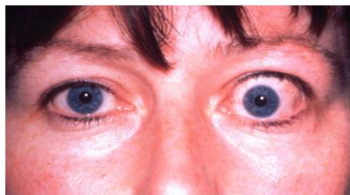


Signs of eyelid retraction

Occurs in about 50%



- Bilateral lid retraction
- No associated proptosis
- Bilateral lid retraction
- Bilateral proptosis

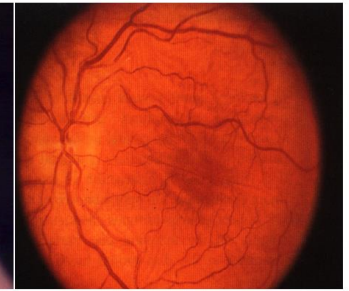


- Unilateral lid retraction
- Unilateral proptosis
- Lid lag in downgaze

- Unilateral proptosis
- Unilateral lid retraction
- Lid lag in downgaze

Proptosis

- Occurs in about 50%
- Uninfluenced by treatment of hyperthyroidism



Axial and permanent in about 70%

May be associated with choroidal folds

- Treatment options**
- Systemic steroids
 - Radiotherapy
 - Surgical decompression

Optic Neuropathy

- Occurs in about 5%
- Early defective colour vision
- Usually normal disc appearance



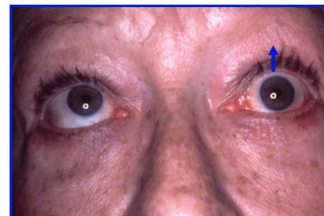
Caused by optic nerve compression at orbital apex by enlarged recti



Often occurs in absence of significant proptosis

Restrictive Myopathy

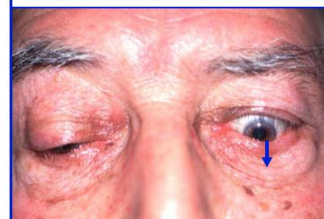
- Occurs in about 40%
- Due to fibrotic contracture



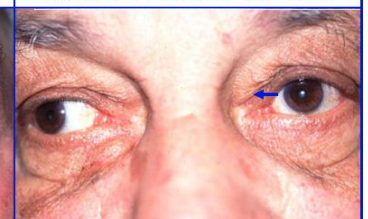
Elevation defect - most common



Abduction defect - less common



Depression defect - uncommon



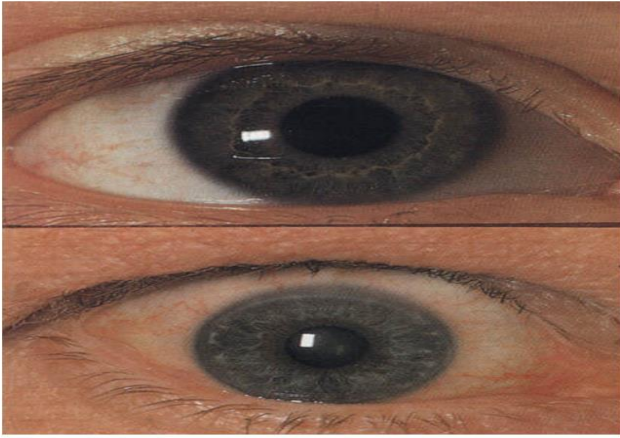
Adduction defect - rare

Depression defect - uncommon

Adduction defect - rare

Thyrotoxicosis

Eyelid retraction



Thyrotoxicosis

Staring and frightened appearance



Thyrotoxicosis



Ocular Manifestations Of HIV Infection

Ophthalmic Manifestations of HIV Infection

A. Around The Eye

1. Molluscum Contagiosum
2. Herpes Zoster Ophthalmicus
3. Kaposi's Sarcoma
4. Conjunctival Squamous Cell Carcinoma
5. Trichomegaly

B. Front Of The Eye

1. Dry Eye
2. Anterior Uveitis

C. Back Of The Eye

1. Retinal Microvasculopathy
2. CMV Retinitis
3. Acute Retinal Necrosis
4. Progressive Outer Retinal Necrosis
5. Toxoplasmosis Retinochoroiditis
6. Syphilis Retinitis
7. Candida albicans endophthalmitis

D. Neuro-Ophthalmic

Human Immunodeficiency Virus

- most of the ocular infections are from various opportunistic organisms , The opportunistic infections include:

1. Cytomegalovirus retinitis
2. Progressive outer retinal necrosis
3. Toxoplasmic retinochoroiditis
4. Fungal chorioretinitis
5. Infectious multifocal choroiditis
6. Molluscum contagiosum.

Viral Infection HIV

Acute retinal necrosis



Human Immunodeficiency Virus

- Neoplasms, which involve the eyelids, conjunctiva, and other ocular and orbital structures.
- Anterior segment diseases, such as herpes simplex, herpes zoster, and ulcerative keratitis,

Progressive Outer Retinal Necrosis

- Progressive outer retinal necrosis (porn) is less common than cytomegalovirus (cmv) retinitis, the former causes a more rapid destruction of the retina and carries a poor prognosis
- Herpes zoster is the causative agent of retinal necrosis.

Viral infection (Herpes zoster)



Progressive Outer Retinal Necrosis

- Characterizes by:
 - deep outer retinal lesions in a circumferential pattern in the peripheral retina
 - These lesions tend to coalesce rapidly and progress to full-thickness retinal necrosis in a matter of days
 - Within a matter of weeks vision may deteriorate from (6/6) to (6/9) to light perception to no light perception

Progressive Outer Retinal Necrosis



Congenital Rubella

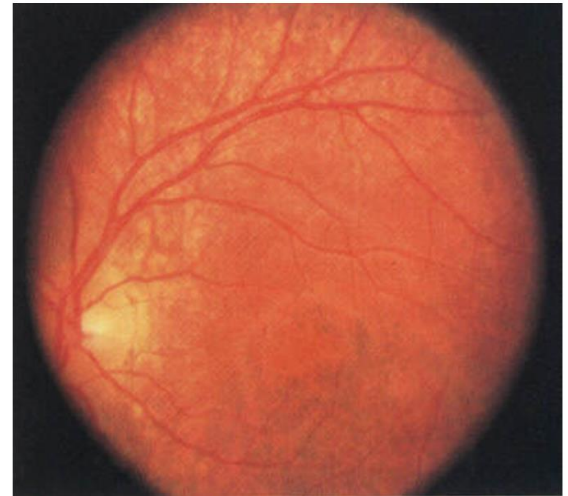
- Congenital rubella results from transplacental transmission of virus to the fetus from an infected mother, usually during the first trimester of pregnancy
- **Systemic complications** of maternal rubella include: spontaneous abortion, stillbirth, congenital heart malformations, deafness, microcephaly, mental handicap, hypotonia, hepatosplenomegaly, thrombocytopenia.
- **OCULAR FEATURES**

Retinopathy

- Is the most common ocular complication
- The characteristic finding is a 'salt and pepper' pigmentary disturbance, most often involving the posterior pole and most marked at the macula
- optic nerve head and retinal blood vessels are usually normal.

Viral Infection (German Measles)

'Salt and pepper' pigmentary disturbance



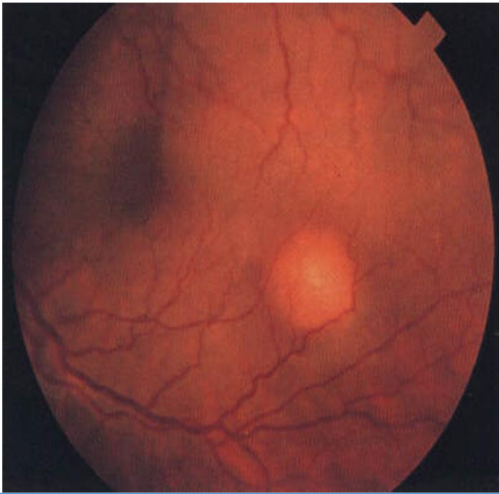
Viral Infection (German Measles)

Congenital Cataract

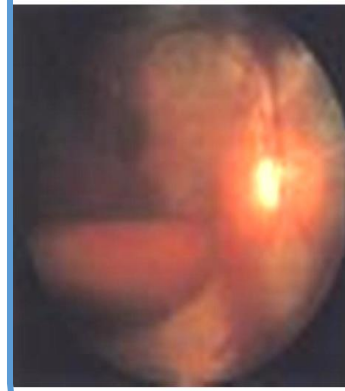


Tuberculosis

Solitary Tuberculous Choroidal Granuloma

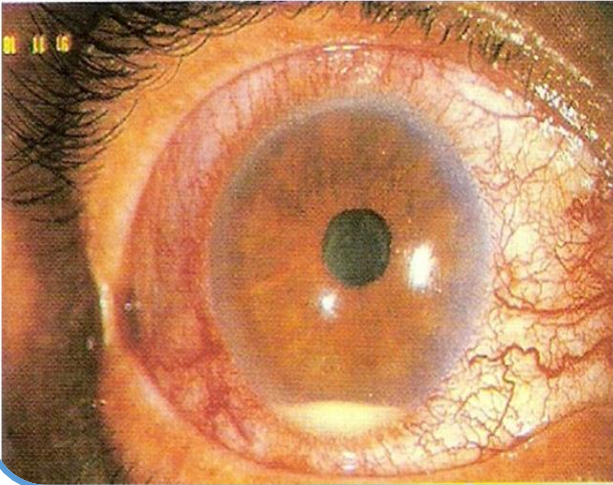


Eael's Disease

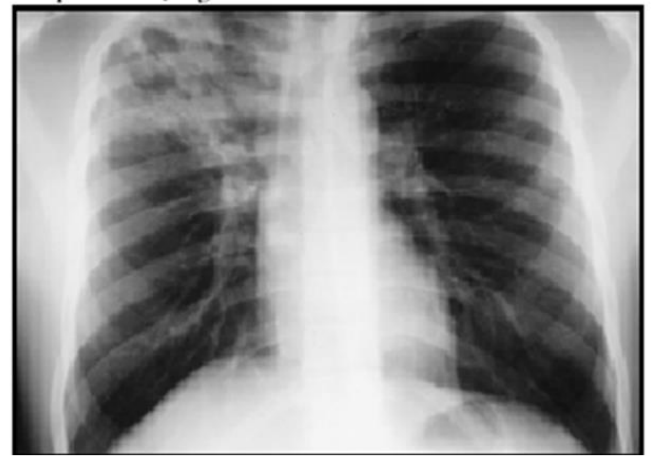


- Vasculitis
- Young patient
- Recurrent of vitreous hemorrhage

Acute TB uveitis with hypopyon, posterior synechia



Chest X Ray For Pul.TB



Ocular TB

Uveitis

Examination:

1. Mutton fat keratic precipitate.
2. Extensive posterior synechia
3. Cataract and secondary glaucoma (long standing)

Syphilis

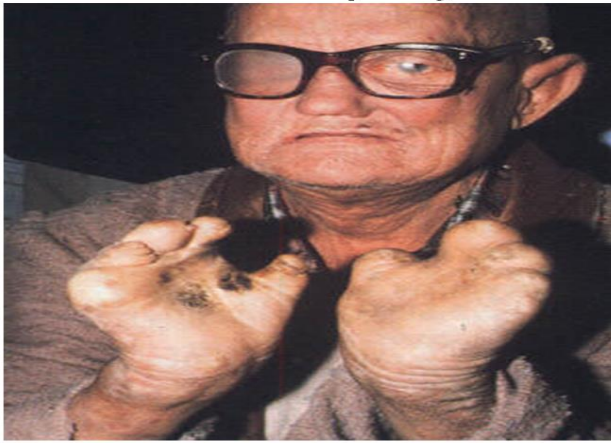
Argyll Robertson Pupil

The Argyll Robertson pupil is caused by neurosyphilis and is characterized by the following:

1. Involvement is usually bilateral but asymmetrical.
2. The pupils are small and irregular.
3. The light reflex is absent or very sluggish.
4. Light near dissociation (The near reflex is normal)
5. The pupils are difficult to dilate.

Leprosy

Advanced Leprosy



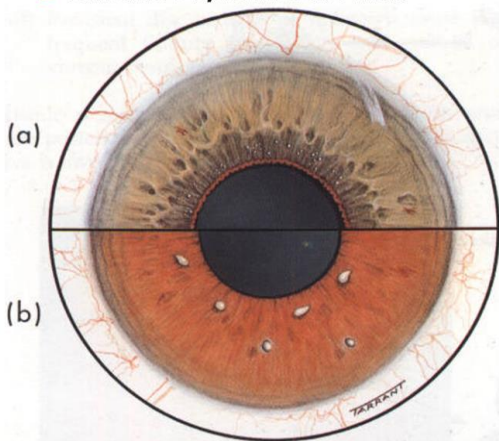
Leprosy

severe corneal scarring, And lagophthalmos



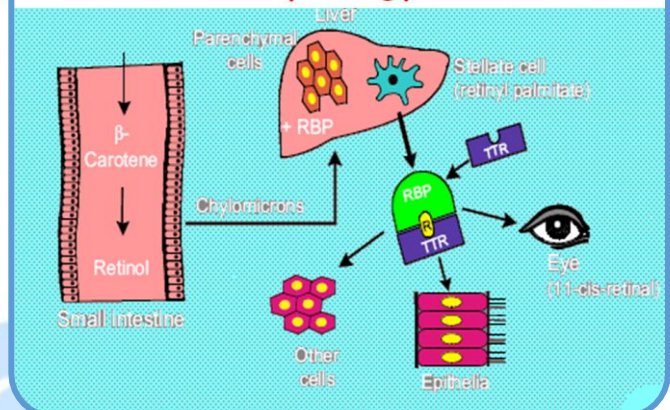
Leprosy

Chronic Lepromatous Iritis



Vitamin A deficiency

Physiology



Major Food Sources



WHO Criteria Of Vitamin A Deficiency

XN	Night blindness
X1A	Conjunctival xerosis
X1B	Bitot's spots
X2	Corneal xerosis
X3A	Corneal ulceration/keratomalacia (involving <1/3 of the corneal area)
X3B	Corneal ulceration/keratomalacia (involving ≥1/3 of the corneal area)
XS	Corneal scar
XF	Xerophthalmic fundus

XF	Xerophthalmic fundus
X2	Corneal scar
X3A	Corneal ulceration/keratomalacia (involving <1/3 of the corneal area)

Conjunctiva

- Conjunctival xerosis→earliest, clinically detectable structural change.
- Bitot's spots→ keratinized epithelial cells which accumulate above the conjunctival surface.



Bitot's Spots (X1B)

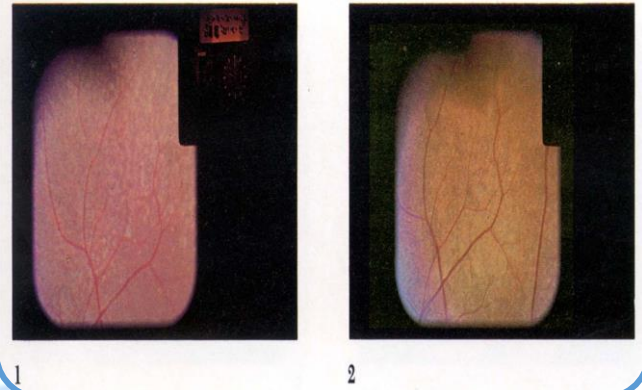


Vitamin A deficiency

Conjunctival Xerosis



Xerophthalmic Fundus (XF)



Cornea

- Corneal Scarring → Corneal Opacities.
- Staphyloma.
- Phthisis Bulbi.



Vitamin A Deficiency

Treatment :

1. More than one year of age :
 - 200,000 IU first day orally
 - 200,000 IU second day orally
 - 200,000 IU 4 weeks later orally
2. Less than one year of age:
 - Half of the dose orally
 - Parentrally: half of the dose
3. Preventive treatment :
 - Less than one year 100,000 IU / 6 months
 - More than one year 200,000 IU / 6 months

Intra-Ocular Tumors

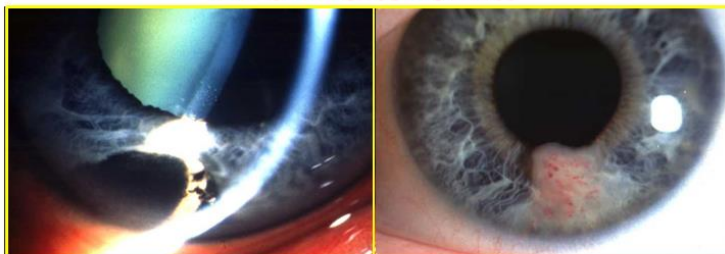
Uveal Tumours

1. Iris melanoma
2. Iris naevus
3. Ciliary body melanoma
4. Choroidal melanoma
5. Choroidal naevus
6. Choroidal haemangioma
 - Circumscribed
 - Diffuse
7. Choroidal metastatic carcinoma
8. Choroidal osseous choristoma
9. Melanocytoma

Iris Melanoma

1. Very rare - 8% of uveal melanomas
2. Presentation - fifth to sixth decades
3. Very slow growth
4. Low malignancy
5. Excellent prognosis

Iris melanoma



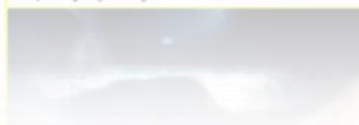
- Usually pigmented nodule at least 3 mm in diameter
- Invariably in inferior half of iris
- Occasionally non-pigmented
- Surface vascularization



- Angle involvement may cause glaucoma
- Pupillary distortion, ectropion uveae and cataract

Glaucoma

• Angle involvement may cause

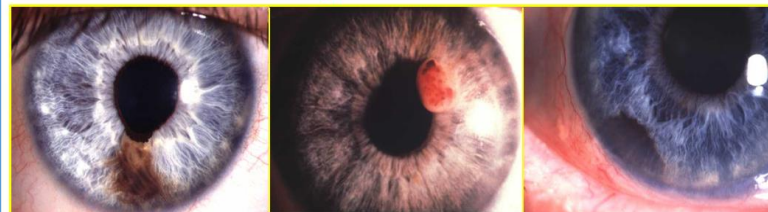


Pupillary distortion, ectropion

• Pupillary distortion, ectropion



Differential diagnosis of iris melanoma



Large iris naevus distorting pupil

Leiomyoma

Adenoma of pigment epithelium

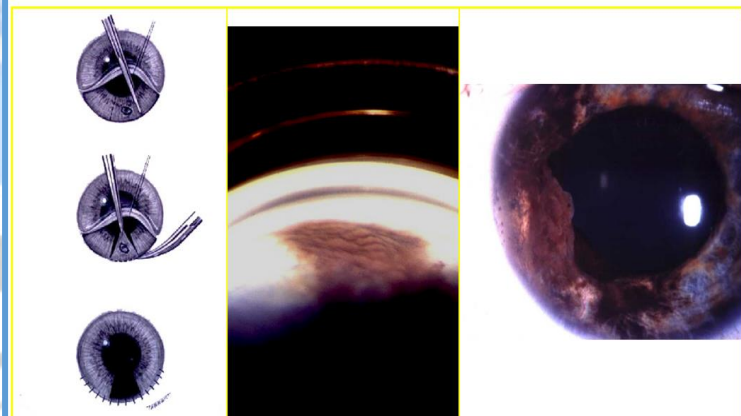


Primary iris cyst

Ciliary body melanoma eroding iris root

Metastasis to iris

Treatment of iris melanoma



Small tumour - broad iridectomy

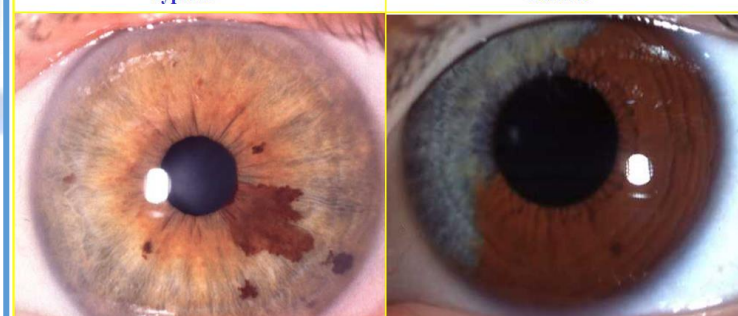
Angle invasion by tumour - iridocyclectomy

Non-resectable tumour - radiotherapy or enucleation

Iris naevus

Typical

Diffuse

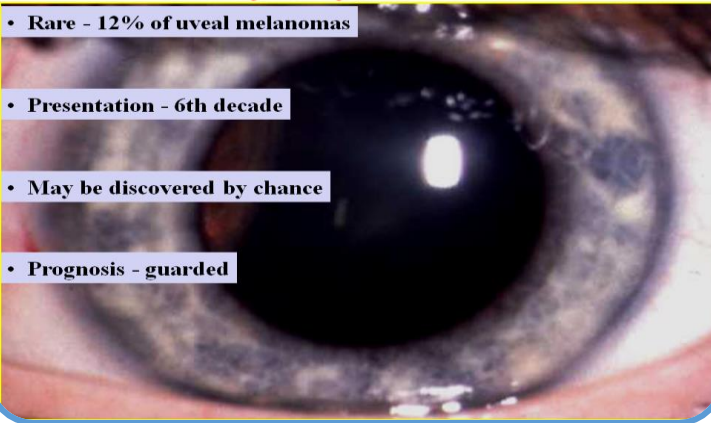


- Pigmented, flat or slightly elevated
- Diameter usually less than 3 mm
- Occasionally mild distortion of pupil and ectropion uvea

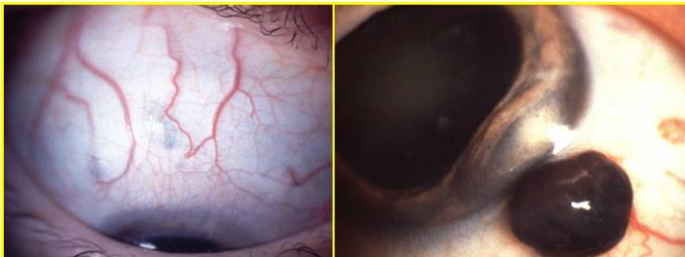
- Obscures iris crypts
- May cause ipsilateral hyperchromic heterochromia
- May be associated with Cogan-Reese syndrome

Ciliary body melanoma

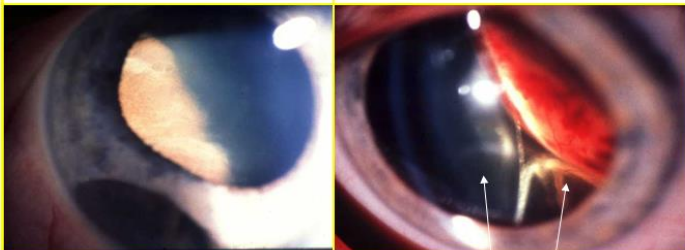
- Rare - 12% of uveal melanomas
- Presentation - 6th decade
- May be discovered by chance
- Prognosis - guarded



Signs of ciliary body melanoma



- Sentinel vessels
- Extraocular extension



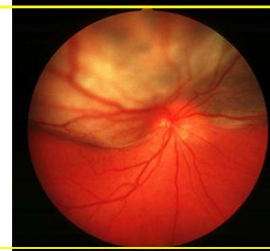
- Erosion through iris root
- Lens subluxation or cataract
- Retinal detachment

Choroidal melanoma

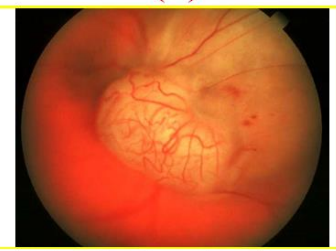
- Most common primary intraocular tumour in adults
- Most common uveal melanoma - 80% of cases
- Presentation - sixth decade
- Prognosis - usually good



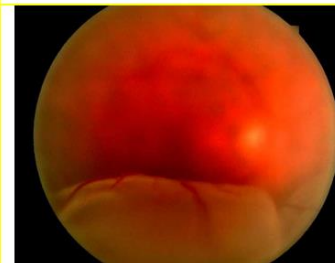
Choroidal Melanoma (1)



- Brown, elevated, subretinal mass



- Occasionally amelanotic
- Double circulation



- Secondary retinal detachment



- Choroidal folds

Choroidal Melanoma (2)



- Surface orange pigment (lipofuscin) is common
- Mushroom-shaped if breaks through Bruch's membrane



- Ultrasound - acoustic hollowiness, choroidal excavation and orbital shadowing

Treatment options of ciliary body melanoma

1. Iridocyclectomy
 - small or medium tumours
2. Enucleation
 - large tumours
3. Radiotherapy
 - selected cases

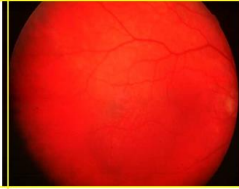
Differential Diagnosis Of Choroidal Melanoma



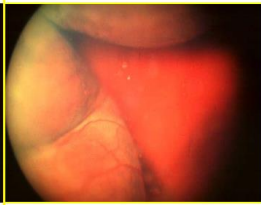
Large choroidal naevus



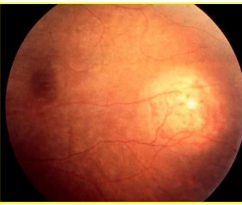
Metastatic tumour



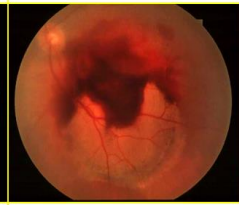
Localized choroidal haemangioma



Choroidal detachment



Choroidal granuloma



Dense sub-retinal or sub-RPE haemorrhage

Poor Prognostic Factors of Uveal Melanomas

1. Histological
 - Epithelioid cells
 - Closed vascular loops
 - Lymphocytic infiltration
2. Large size
3. Extrascleral extension
4. Anterior location
5. Age over 65 years

Typical Choroidal Naevus



Common - 2% of population

Round slate-grey with indistinct margins

Surface drusen

Flat or slightly elevated

Diameter less than 5 mm

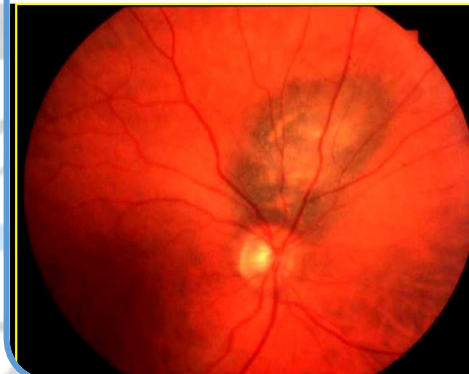
Location - anywhere

Asymptomatic

Treatment of choroidal melanoma

1. Brachytherapy
 - less than 10 mm elevation and 20 mm diameter
2. Charged particle irradiation
 - if unsuitable for brachytherapy
3. Transpupillary thermotherapy
 - selected small tumours
4. Trans-scleral local resection
 - carefully selected tumours less than 16 mm in diameter
5. Enucleation
 - very large tumours, particularly if useful vision lost
6. Exenteration
 - extraocular extension

Suspicious Choroidal Naevus



• Diameter more than 5 mm

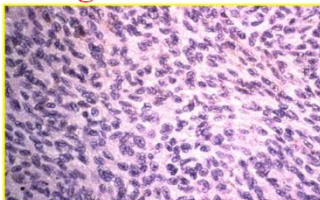
• Elevation 2 mm or more

• Surface lipofuscin

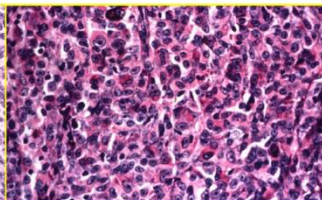
• Posterior margin within 3 mm of disc

• May have symptoms due to serous fluid

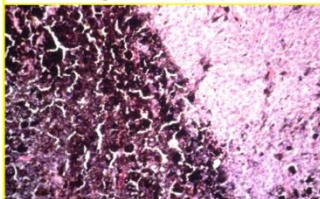
Histological Classification Of Uveal Melanomas



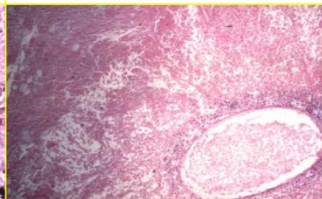
Spindle cell (45%)



Pure epithelioid cell (5%)

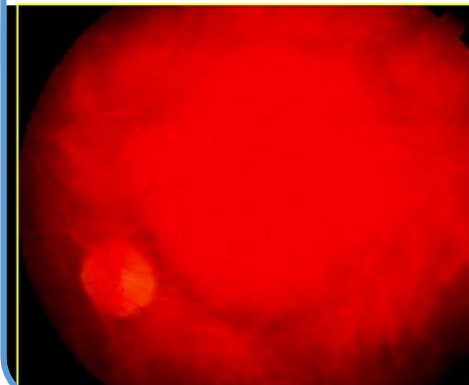


Mixed cell (45%)



Necrotic (5%)

Circumscribed Choroidal Haemangioma



• Presentation - adult life

• Dome-shaped or placoid, red-orange mass

• Commonly at posterior pole

• Between 3 and 9 mm in diameter

• May blanch with external globe pressure

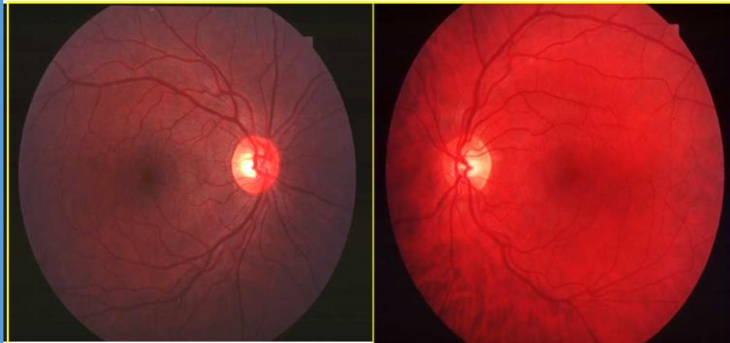
• Surface cystoid retinal degeneration

• Exudative retinal detachment

• Treatment - radiotherapy if vision threatened

Diffuse Choroidal Haemangioma

Typically affects patients with Sturge-Weber syndrome

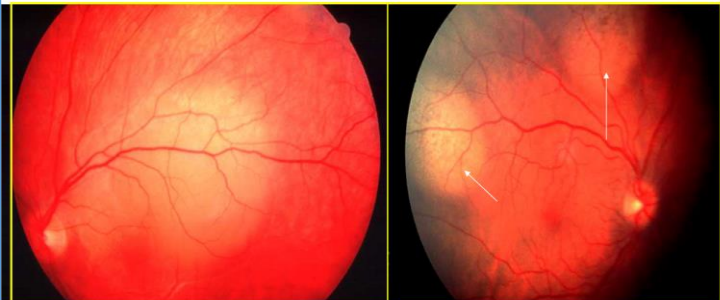


Can be missed unless compared with normal fellow eye as shown here

Diffuse thickening, most marked at posterior pole

Choroidal Metastatic Carcinoma

Most frequent primary site is breast in women and bronchus in both sexes



- Fast-growing, creamy-white, placoid lesion
- Most frequently at posterior pole

- Deposits may be multiple
- Bilateral in 10-30%

Choroidal Osseous Choristoma



Very rare, benign, slow-growing ossifying tumour

Typically affects young women

Orange-yellow, oval lesion

Well-defined, scalloped, geographical borders

Most commonly peripapillary or at posterior pole

Diffuse mottling of RPE

Bilateral in 25%

Melanocytoma



- Affects dark skinned individuals
- Usually asymptomatic
- Most frequently affects optic nerve head
- Black lesion with feathery edges

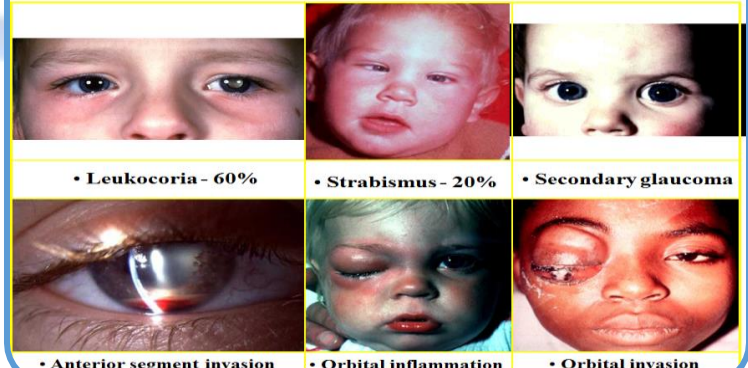
Retinoblastoma

1. Important facts
2. Presentation
3. Signs
 - Endophytic
 - Exophytic
4. Treatment
5. Poor prognostic factors
6. Histology
7. Differential diagnosis of leukocoria

Important Facts

1. Most common primary, malignant, intraocular tumour of childhood (1:20,000)
2. No sexual predilection
3. Presents before age of 3 years (average 3 months)
4. Heritable (40%) or non-heritable (60%)
5. Predisposing gene (RPE 1) on 13q14

Presentations Of Retinoblastoma



• Leukocoria - 60%

• Strabismus - 20%

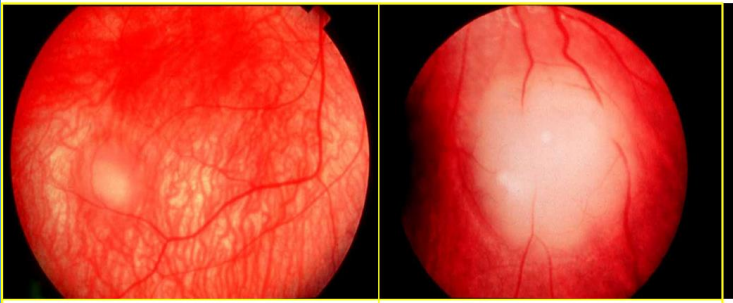
• Secondary glaucoma

• Anterior segment invasion

• Orbital inflammation

• Orbital invasion

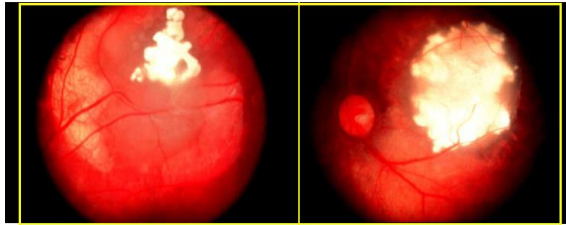
Early Endophyliticretinoblastoma



White flat lesion

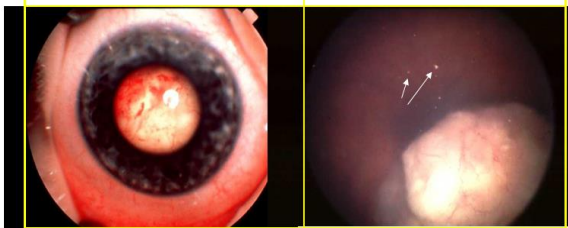
Placoid lesion

More Advanced Endophytic Retinoblastoma



Friable white mass

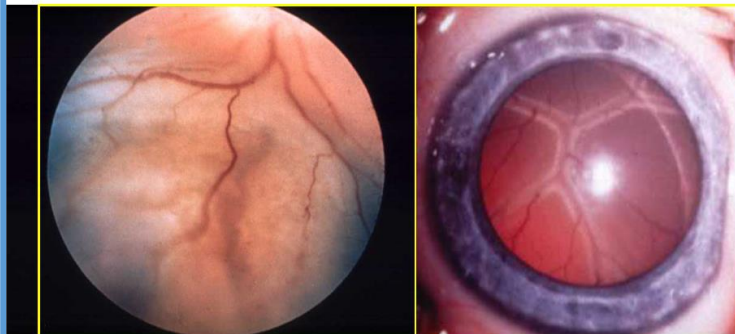
Cottage cheese appearance



Fine surface blood vessels

Vitreous seedings

Exophytic Retinoblastoma



Multiglobulated white mass with overlying retinal detachment

May be difficult to visualize through deep detachment

CT Diagnosis Of Retinoblastoma

Calcification



- Optic nerve involvement
- Orbital and CNS extension
- Pinealoblastoma

- Pinealoblastoma
- Orbital and CNS extension
- Optic nerve involvement

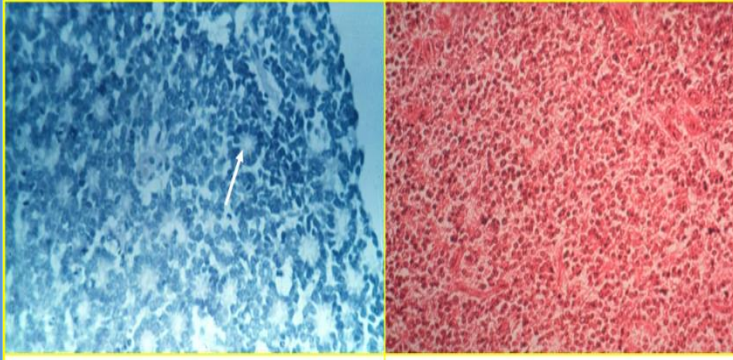
Treatment Options of Retinoblastoma

1. Small tumours
 - Laser photocoagulation
 - Transpupillary thermotherapy
 - Cryotherapy
2. Medium tumours
 - Brachytherapy
 - Chemotherapy
 - External beam radiotherapy
3. Large tumours
 - Chemotherapy followed by local treatment
 - Enucleation
4. Extraocular extension
 - External beam radiotherapy
5. Metastatic disease
 - Chemotherapy

Poor Prognostic Factors in Retinoblastoma

1. Optic nerve involvement
2. Choroidal invasion
3. Large tumour
4. Anterior location
5. Poor cellular differentiation
6. Older children

Histology Of Retinoblastoma



Well-differentiated with many Flexner-Wintersteiner rosettes

Poorly differentiated

Differential Diagnosis Of Leukocoria

Congenital cataract	Persistent hyperplastic primary vitreous	Inflammatory cyclitic membrane
Unilateral or bilateral	Unilateral	Unilateral or bilateral
Coats disease	Posterior pole toxocara granuloma	Advanced retinopathy of prematurity
Unilateral	Unilateral	Always bilateral but may be asymmetrical

Neuro-Ophthalmology

Mahfouth Bamashmus FRCSEd FRCOphth
Professor of Ophthalmology
SANA'A UNIVERSITY



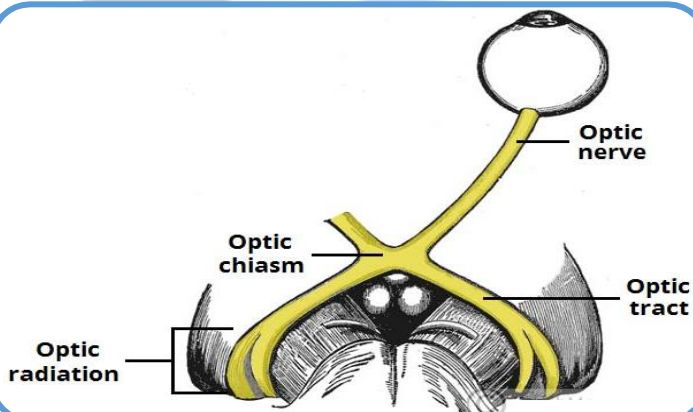
Neuro-Ophthalmology

1. Optic Nerve diseases
 - Papilloedema
 - Optic Atrophy
 - Optic Neuritis
2. Affection of Visual Pathway
3. Affection of Intrinsic Muscles (Pupil)
4. Affection of Extra-Ocular Muscles
5. Others
 - Migraine
 - Myasthenia Gravis

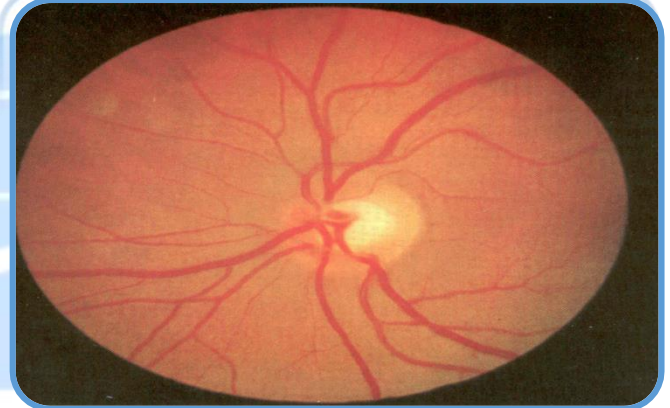
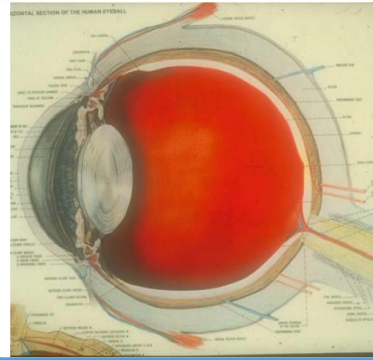
Optic Nerve Diseases

The Visual Pathway

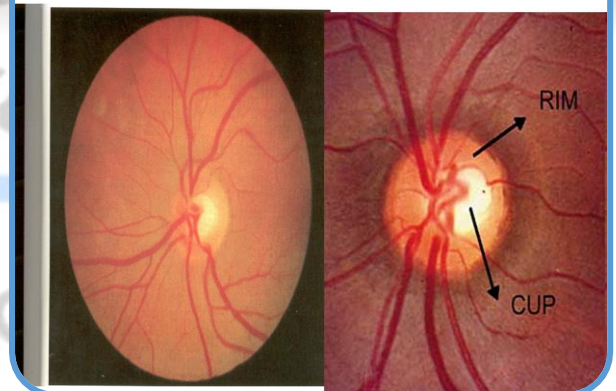
1. The Retina
2. The optic nerve
3. The optic chiasm
4. The optic tract
5. The lateral geniculate body
6. The optic radiation
7. The occipital cortex
 - a) Area 17 (visuo-sensory)
 - b) Areas 18 and 19 (visuo-psychic)



Normal Eye and Optic Disc



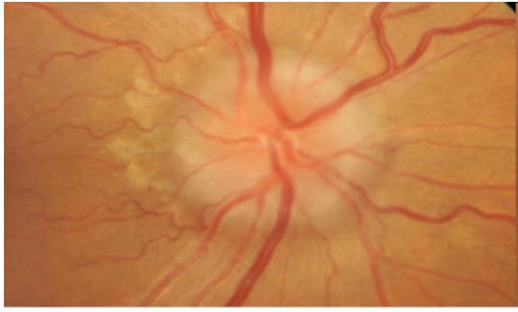
Normal optic disc



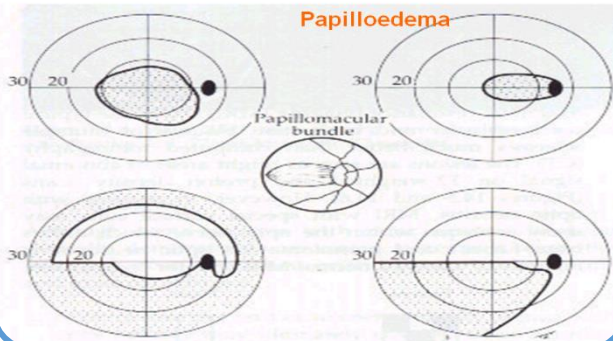
Papilloedema

- Is passive edema of the optic disc which is not accompanied by inflammation or loss of vision.

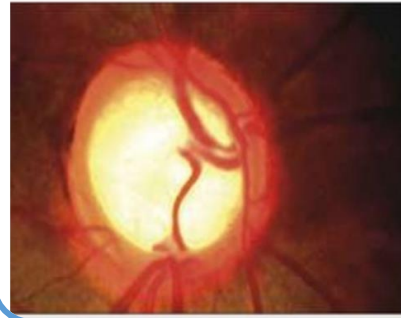
Papilloedema



Visual field defects in Papilledema (Enlarged blind spot)

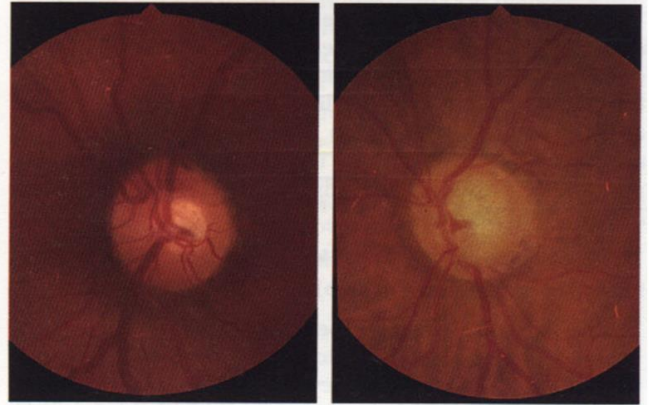


Glaucomatous Optic Atrophy



- Pale optic disc
- Glaucomatous cupping

Glaucomatous Optic Atrophy



Optic Atrophy

- Is optic nerve degeneration

Optic Atrophy



- Milky white color
- well defined edge
- Enlarged physiological cup (atrophic cup)

Optic Neuritis

- Is inflammation of optic disc



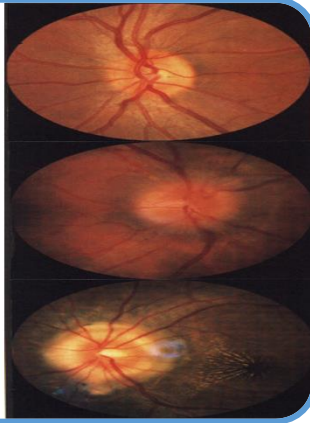
Remember

Optic Nerve

- Anatomy
- Parts of optic nerve
- Diseases of Optic Nerve
 - ☐ Papilloedema
 - ☐ Optic Atrophy
 - ☐ Optic Neuritis

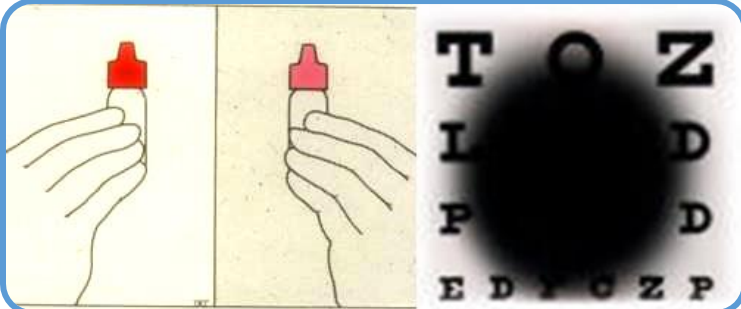
Classification of Optic neuritis

- Retrobulbar neuritis - normal disc
- Papillitis
- Neuroretinitis

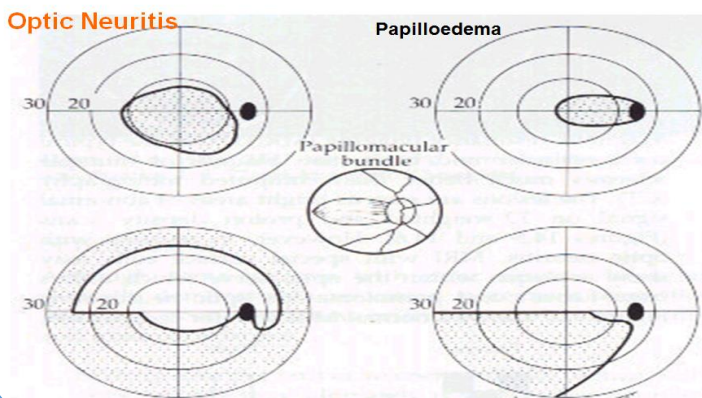


Clinical Examination

1. Visual Acuity (Decreased)
2. Colour Vision (Red colour Desaturation)
3. Visual Fields (central Scotoma)
4. Pain with eye movement
5. Pupils (RAPD)



Visual field defects in optic nerve disease



The swollen optic disc

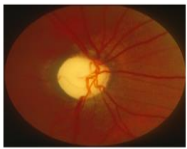
- Papilloedema
- Papillitis
- Malignant hypertension
- Ischaemic optic neuropathy
- Diabetic optic neuropathy
- CRVO
- Intraocular inflammation



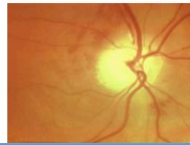
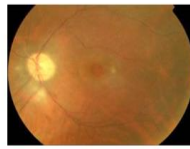
The Pale Optic Disc

1. Primary
2. Secondary to
 - i. raised ICP
 - ii. vascular retinal disease
 - iii. optic neuritis
 - iv. optic nerve compression
3. Consecutive
4. Glaucoma

The pale optic disc



- Primary
- Secondary to
 - raised ICP
 - vascular retinal disease
 - optic neuritis
 - optic nerve compression
- Consecutive
- Glaucoma

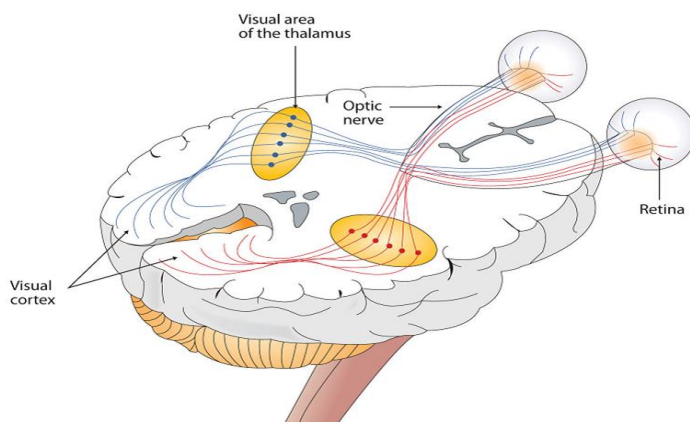
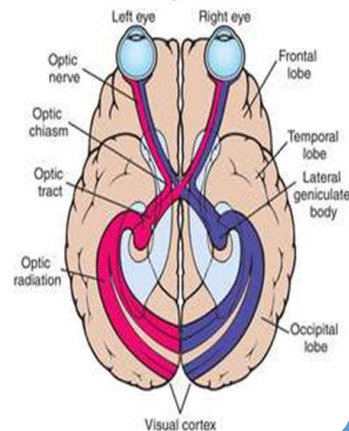


- Glaucoma
- Consecutive

Affection of Visual Pathway

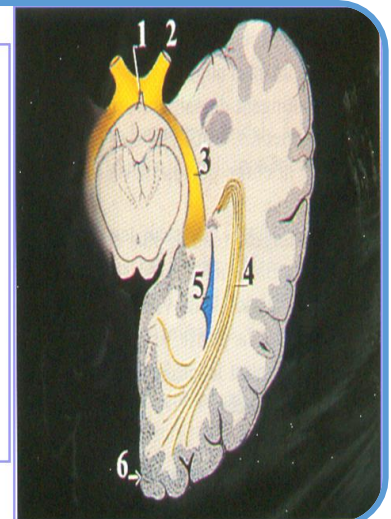
The Visual Pathway

1. The Retina
2. The optic nerve
3. The optic chiasm
4. The optic tract
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6. The optic radiation
7. The occipital cortex
 - a) Area 17 (visuo-sensory)
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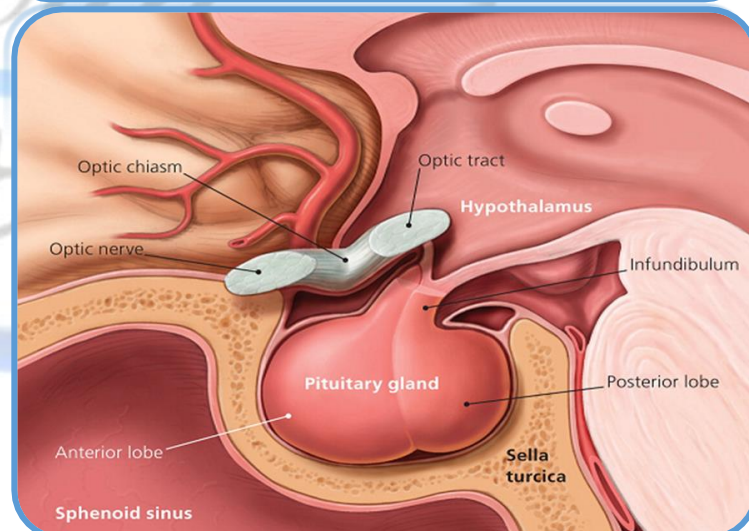
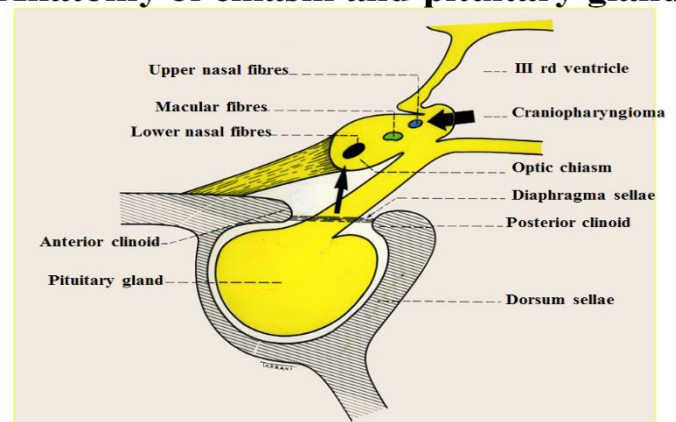


The Optic Chiasm

- Optic chiasm is situated **at junction of anterior wall and floor of 3rd ventricle**
- Fibers from **nasal ½** of each eye **cross to opposite side**
- Fibers from **temporal ½** of each eye **cross directly into optic tract**



Anatomy of chiasm and pituitary gland



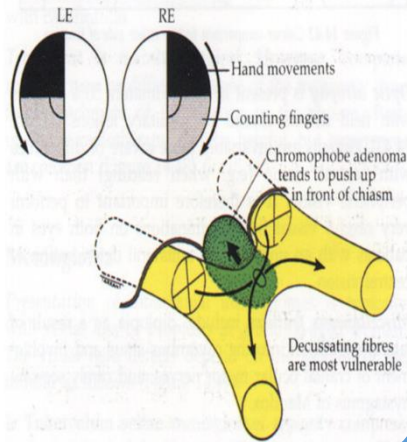
Disorders Of The Chiasm

1. Pituitary adenomas
 - i. Basophil adenoma
 - ii. Acidophil adenoma
 - iii. Chromophobe adenoma
2. Craniopharyngioma
3. Meningioma

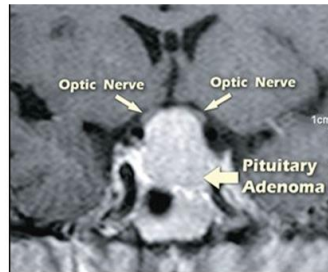
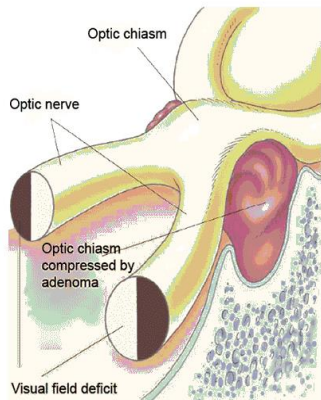
Pituitary Adenoma

Presentation

1. Headache
2. Visual symptoms

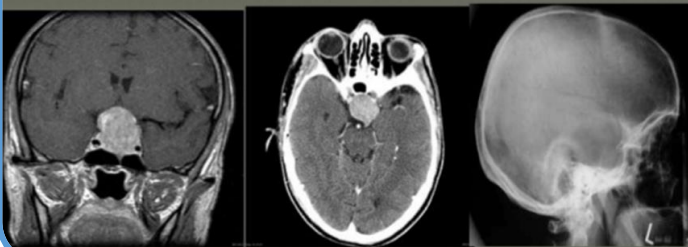


Pituitary Adenoma



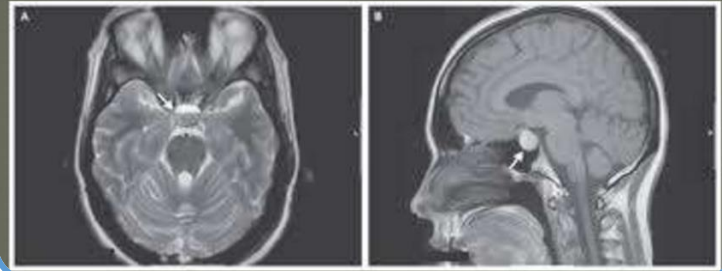
Imaging

- MRI / CT scan of brain
- X-ray skull (Ant. & Lat. view)



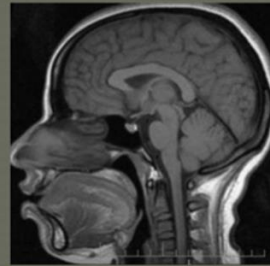
Pituitary Adenoma

Benign tumors of pituitary gland

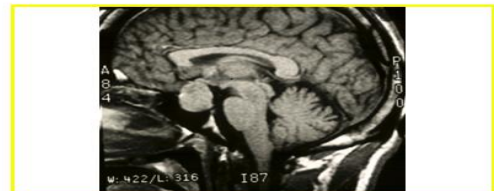


Classification

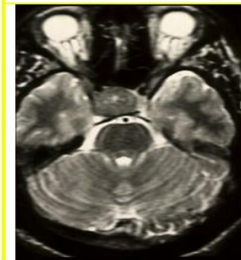
Microadenoma	< 10 mm diameter	Secreting adenoma
Macroadenoma	> 10 mm diameter	Mass effects Non secreting



MRI of pituitary adenoma



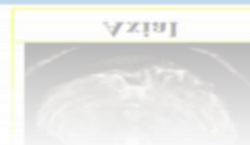
Sagittal



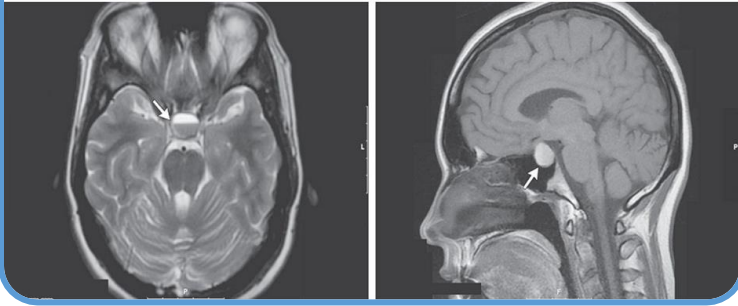
Axial



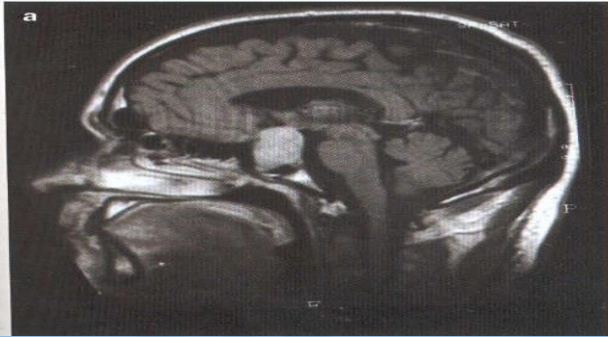
Coronal



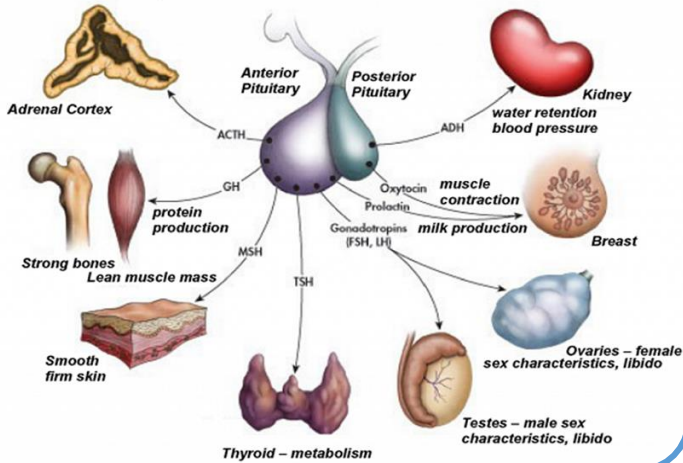
MRI of pituitary adenoma



MRI of pituitary adenoma

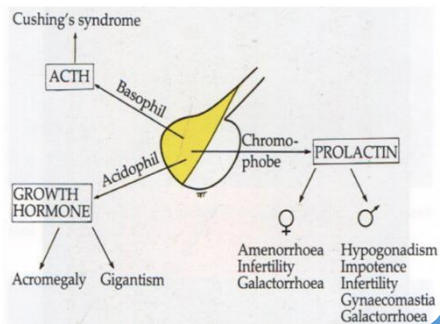


salt & water balance, blood pressure, blood sugar levels, muscle strength, mood, immune system, heart, lungs, blood vessels, nervous system



Disorders of the Chiasm

Hyperpituitarism



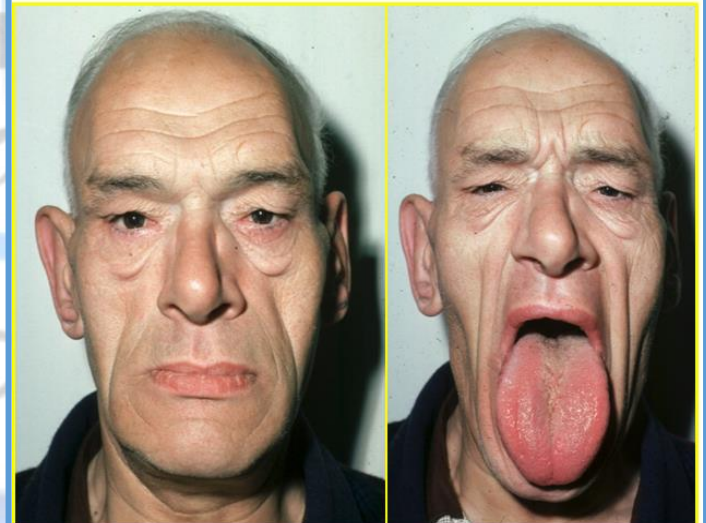
Cushing syndrome



- Moon face, pigmentation and hirsutism
- Hypertension and diabetes

- Obesity, skin striae, bruising and muscle weakness
- Ankle oedema and osteoporosis

Acromegaly



- Facial coarseness
- Hypertension, diabetes and gonadal dysfunction

- Organomegaly
- Carpal tunnel syndrome and cardiomyopathy

Acromegaly



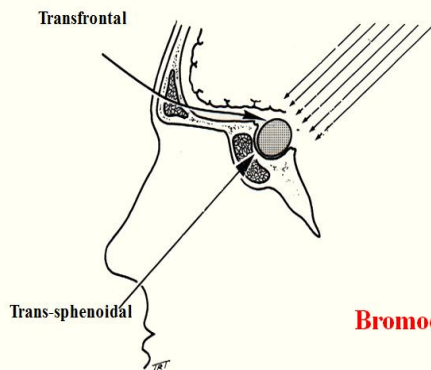
Enlargement of hands and feet



Enlargement of lower jaw

Treatment options for pituitary adenomas

Surgery



Radiotherapy

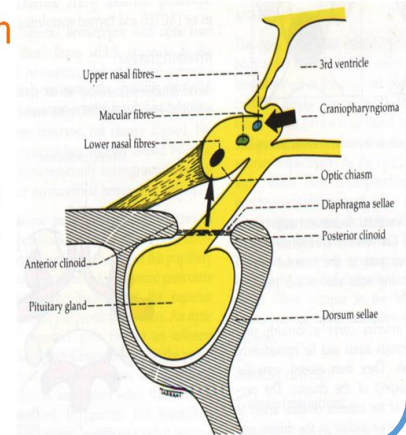
Bromocriptine

Disorders of the Chiasm

■ Hypopituitarism

□ Causes

1. Direct pressure
2. Vascular damage (pituitary apoplexy)
3. Pituitary surgery or radiotherapy



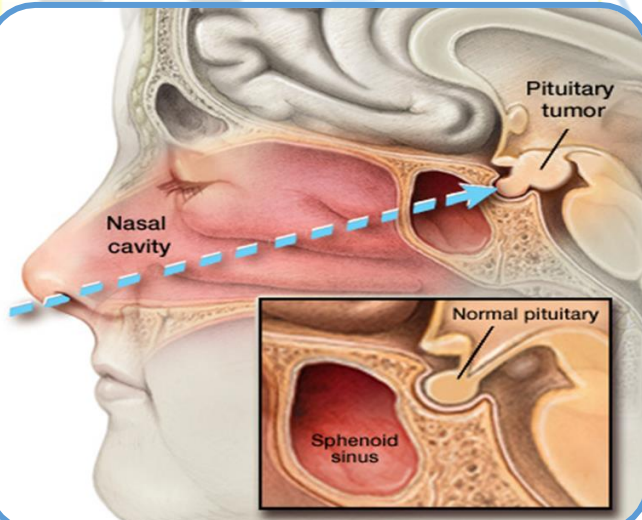
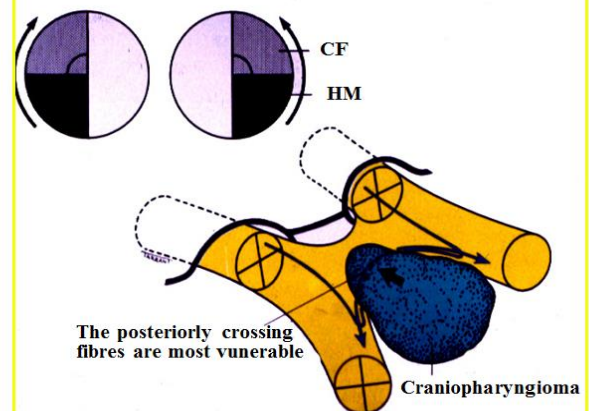
Disorders Of The Chiasm

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2. Craniopharyngioma
3. Meningioma

Craniopharyngioma

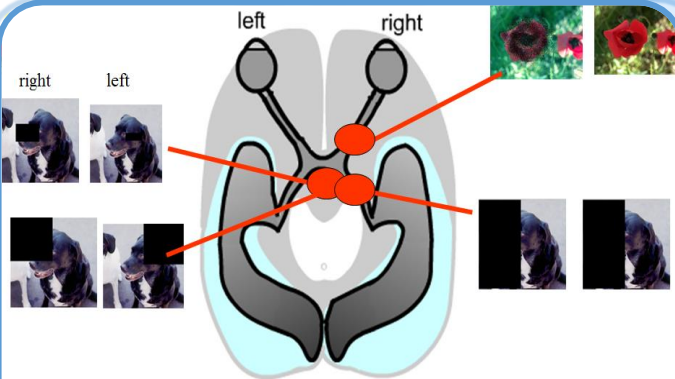
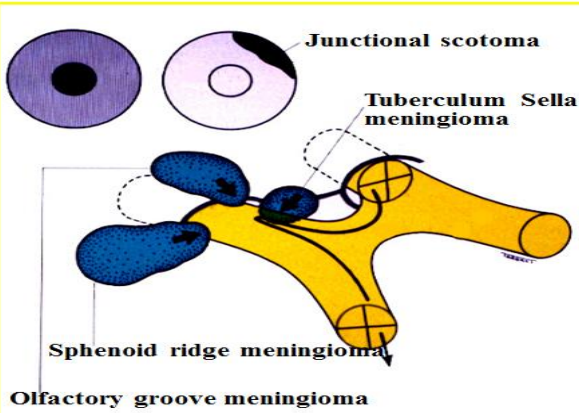
Presents

- In children with endocrine dysfunction
- In adults with visual field defects



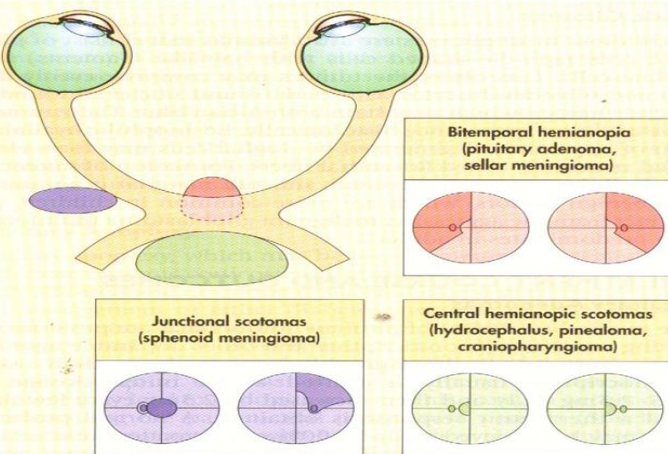
Meningioma

Typically affect middle-aged women



Pituitary (chiasmal) area

LOCALIZATION AND IDENTIFICATION OF MASSES BY PATTERN OF FIELD LOSS



The Visual Pathway

1. The Retina
2. The optic nerve
3. The optic chiasm
4. The optic tract
5. The lateral geniculate body
6. **The optic radiation**
 1. Area 17 (visuo-sensory)
 2. Areas 18 and 19 (visuo-psychic)
7. The occipital cortex

■ Optic Nerve

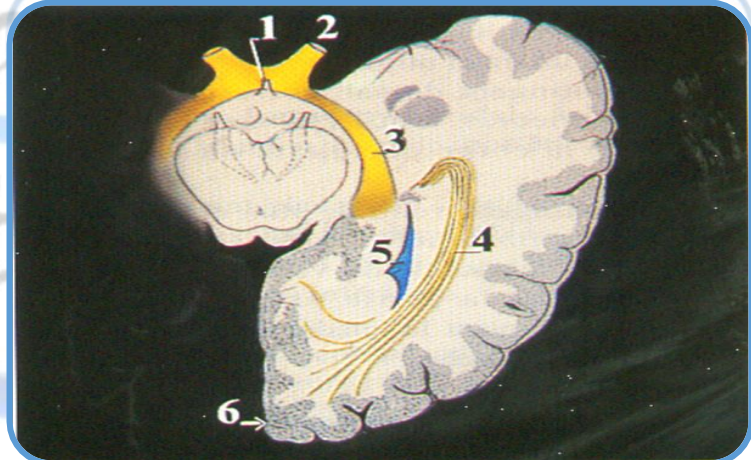
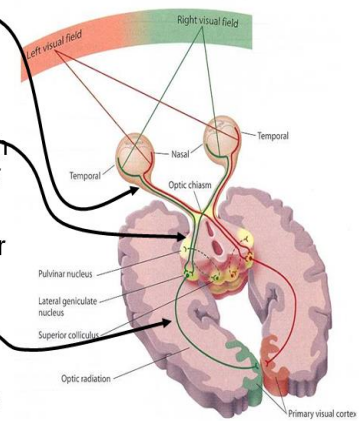
- Visual fibers from retina to optic chiasm

■ Optic Tract

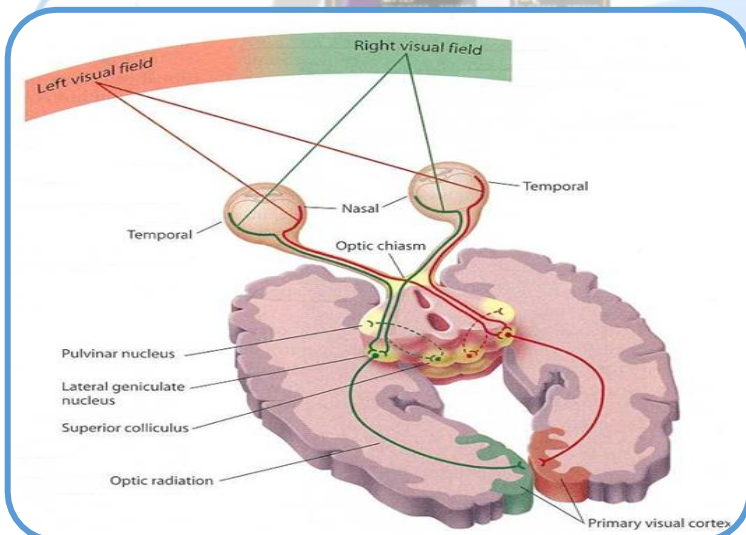
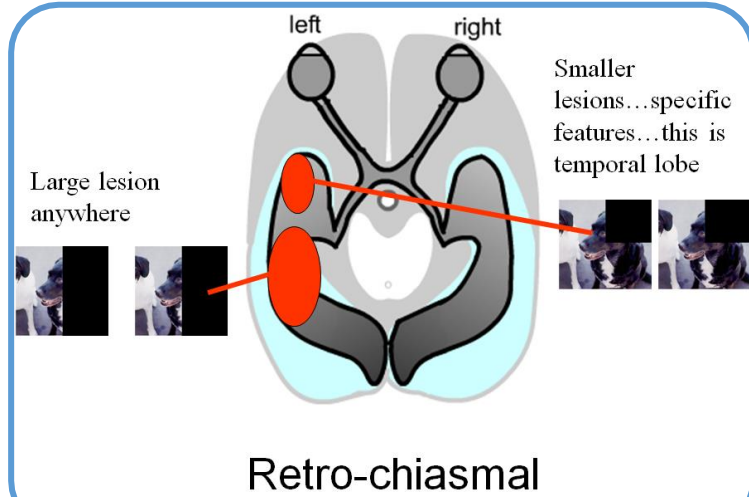
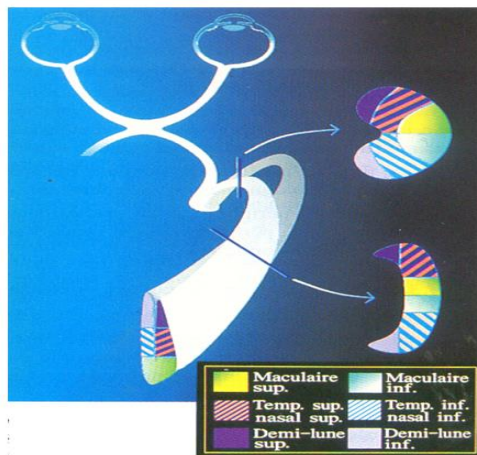
- Optic fibers between chiasm to lateral geniculate body of thalamus or fibers that bypass thalamus to superior colliculus

■ Optic Radiation

- Fibers project to visual cortex via geniculocalcarine fibers (optic radiation)

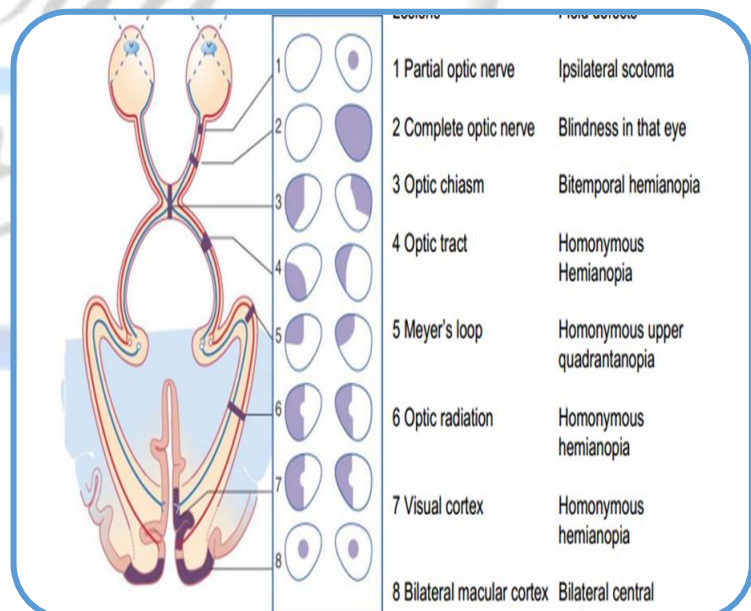
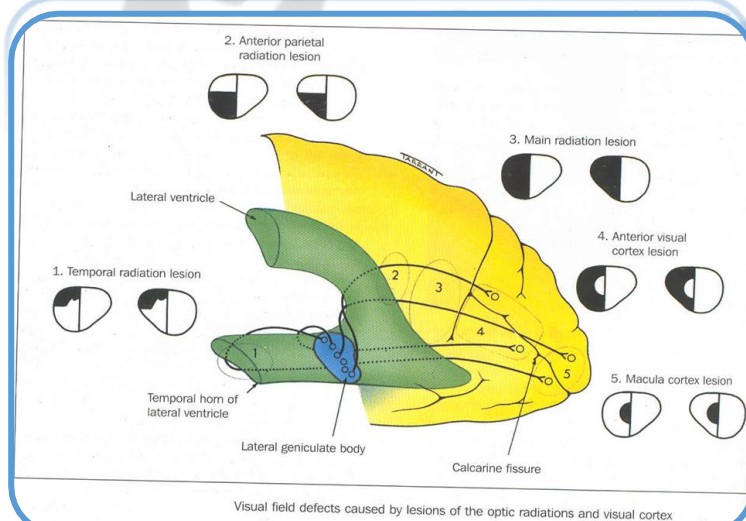


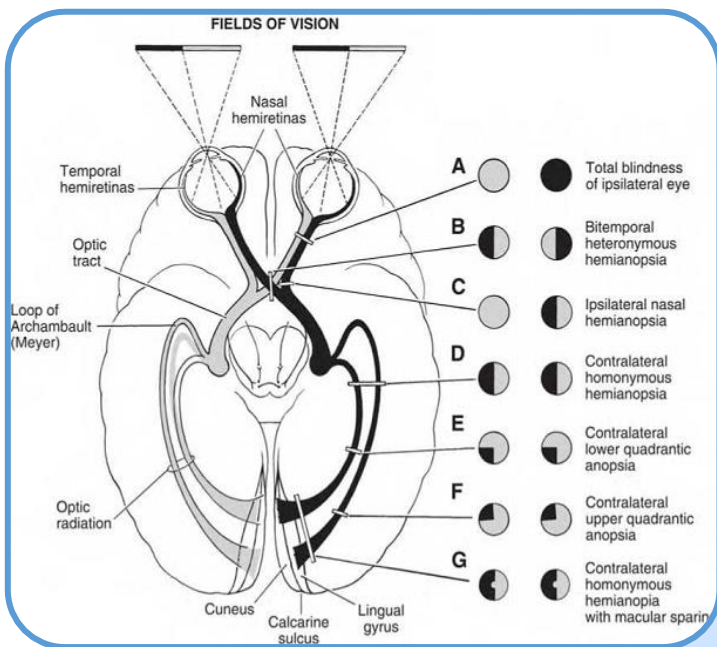
The Optic Radiation



Localising The Lesion

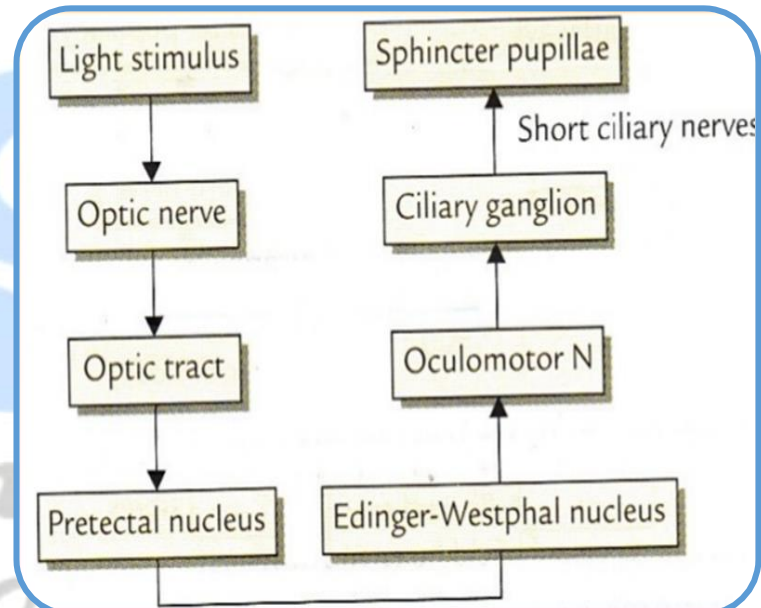
1. Monocular visual field defects indicate lesions anterior to the optic chiasm
2. Bitemporal defects are the hallmark of chiasmal lesions
3. Binocular homonymous hemianopia result from lesions in the contralateral postchiasmal region
4. Binocular quadrantanopias reflect optic tract lesions





Pupils

1. First Order – Retina to Pretectal Nucleus in B/S (at level of Superior colliculus)
2. Second Order – Pretectal nucleus to E/W nucleus (bilateral innervation!)
3. Third Order – E/W nucleus to Ciliary Ganglion
4. Fourth Order – Ciliary Ganglion to Sphincter pupillae (via short ciliary nerves).

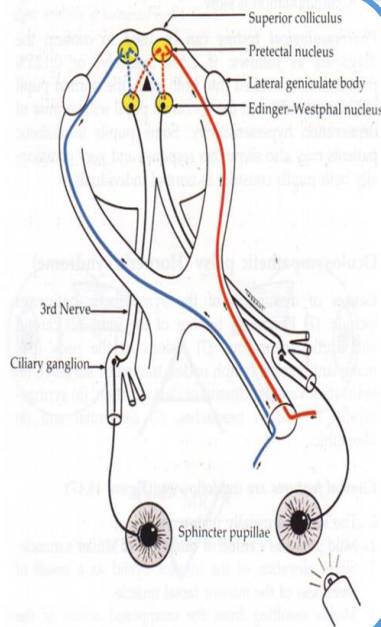


Affection of Intrinsic Muscles (Pupil)

Pupillary Reactions

Parasympathetic

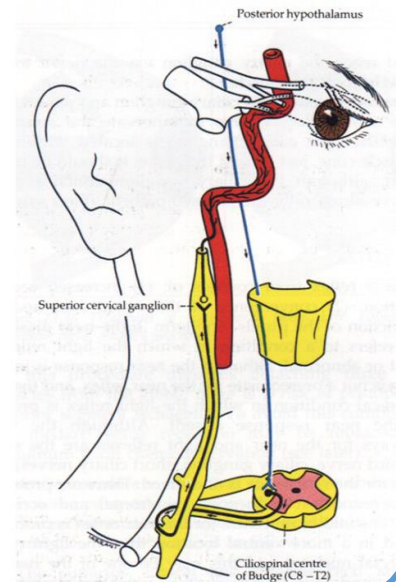
- First Neurone
- Second Neurone
- Third Neurone
- Fourth Neurone



Pupillary Reactions

Sympathetic

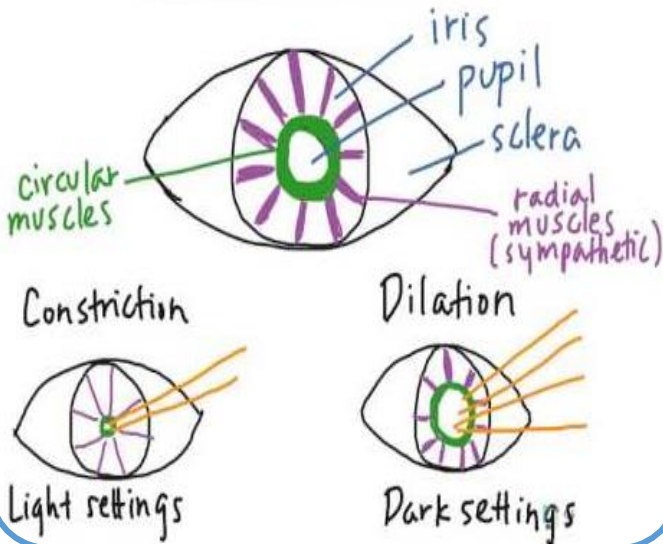
- First Neurone
- Second Neurone
- Third Neurone



Sympathetic Pathway

1. First Order – Posterior Hypothalamus to Ciliospinal centre of Budge (C8-T2) (Uncrossed in Brainstem)
2. Second Order – Ciliospinal centre of Budge to Superior Cervical Ganaglion.
3. Third Order – Superior Cervical Ganglion to dilator pupillae muscle. (Close to ICA and joins V₁ intracranially).

Pupillary Light Reflex



HUMAN EYE (size of the pupil)



In the dark

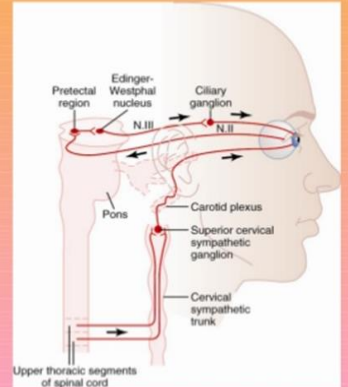


In a brightly lit place

Pupillary Light Reflex

Pupillary Light Reflex

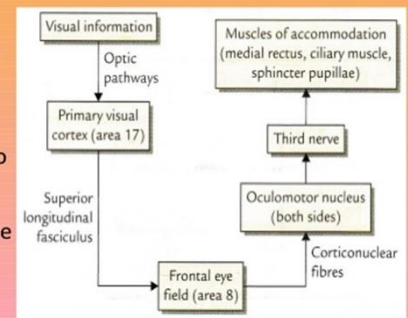
- When the amount of light entering the eyes increases, the pupils constrict.
- Functions to help the eye adapt extremely rapidly to changing light conditions.
- **Direct light reflex:** same pupil constricts
- **indirect (consensual) light reflex:** pupil of the opposite eye constricts



Pupillary Near Reflex

ACCOMMODATION REFLEX

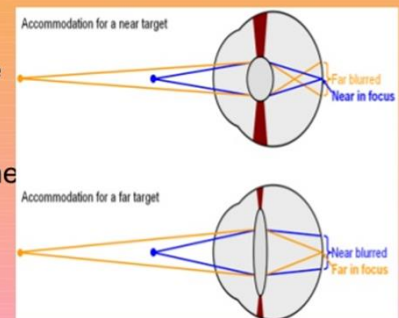
- When the eyes are focussed from a distant to near object, three reactions take place
 - 1. Constriction of pupils
 - 2. thickening of lens due to contraction of ciliary muscles
 - 3. Convergence of both eye balls
- These three reactions together constitute Accommodation or near reflex



Pupillary Near Reflex

Accommodation

- It is the ability of the eye to keep the image focused on the retina (as the distance between the eyes & the object varies)

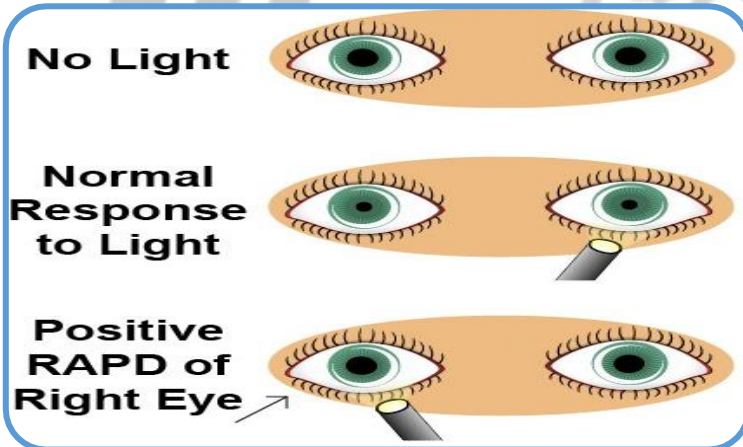


Pupil

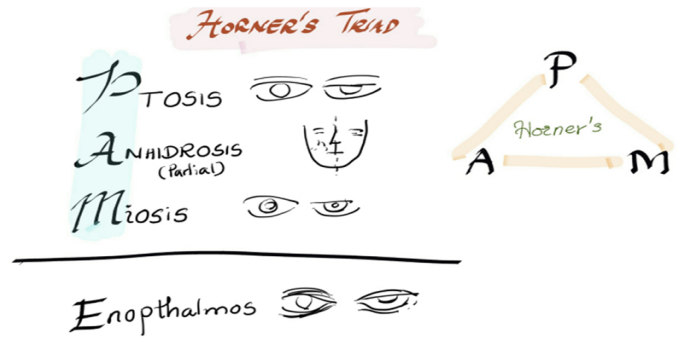
- A. Constricted (*miosis*)
 - 1. Sympathetic (*pupillodilator*) denervation
 - 2. Drugs
 - a. Pilocarpine
 - b. Morphine
- B. Dilated (*mydriasis*)
 - 1. Parasympathetic (*pupilloconstrictor*) denervation
 - 2. Lesion of the third CN
 - 3. Drugs
 - a. Atropine
 - b. Tropicamide
 - c. Cyclopentolate
 - d. Cocaine

Abnormal Pupillary Reaction

- 1) Afferent pupillary conduction defect
- 2) Horner syndrome
- 3) Argyll Robertson pupils
- 4) Adie pupil



Horner's syndrome Signs



Horner's Syndrome Oculo-sympathetic Palsy



Horner's syndrome Signs

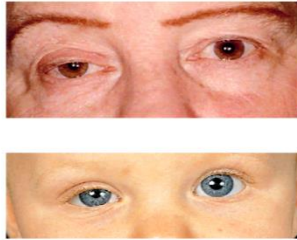
- 1. Mild Ptosis
- 2. Miosis
- 3. Reduced ipsilateral sweating (Anhidrosis)

Other Findings

- Heterochromia
- The pupil is slow to dilate

Horner's Oculosympathetic paresis

- Ptosis
- Miosis
- Ipsilateral anhidrosis



Ptosis (drooping of the eyelid)



Congenital Horner's Syndrome



Left pupillary miosis, marked hypochromia of the left iris, ipsilateral mild ptosis and left hemifacial anhidrosis

Congenital Horner's Syndrome

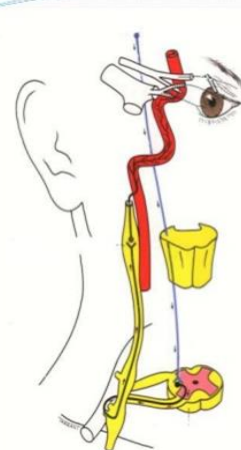


Causes Of Disruption Of Sympathetic Pathways

1. Pancoast's Tumor of the lung
2. Carotid and aortic aneurysms
3. Lesions of the neck (eg Malignant cervical LN, trauma, surgery)
4. Brain-stem vascular disease or demyelination
5. Cluster Headache
6. Congenital

Causes of disruption of Sympathetic pathways

Important causes of Horner syndrome



Central
(first order neurone)

- Brainstem disease (vascular, demyelination)
- Spinal cord disease (syringomyelia, tumours)

Pre-ganglionic
(second order neurone)

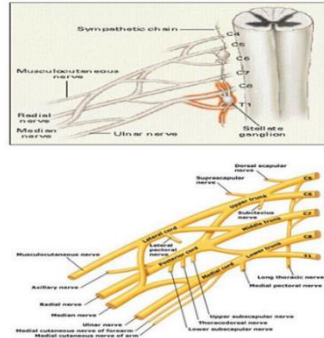
- Intrathoracic lesions (Pancoast tumour, aneurysm)
- Neck lesions (glands, trauma)

Post-ganglionic
(third order neurone)

- Internal carotid artery disease
- Cavernous sinus mass

Pancoast tumor

- **Right Shoulder Pain**
 - Commonest first symptom (44-96%)
 - invasion into brachial plexus, 1st or 2nd ribs, Vertebral bodies, parietal pleura, chest wall
- **Horner's Syndrome**
 - 14-50%.
 - Paravertebral sympathetic chain and inferior stellate ganglion
- **Neurological complication of the upper limbs**
 - C8/T1 Nerve root involvement
- **10% profound weight loss**
- **10% have Recurrent laryngeal nerve palsy**



Raeder's syndrome

- Horner's with pain in the distribution area of V1.
- Caused by a neoplasm compressing the trigeminal nerve.
- Differential for cluster headaches.



Argyll Robertson Pupils

- Primary gaze



- Light response



- Near response



Argyll Robertson pupils:

Caused by neurosyphilis.

They are characterized by bilateral (usually asymmetrical) small, irregular pupils showing a light-near dissociation.

Difficult to dilate.



Adie Pupil

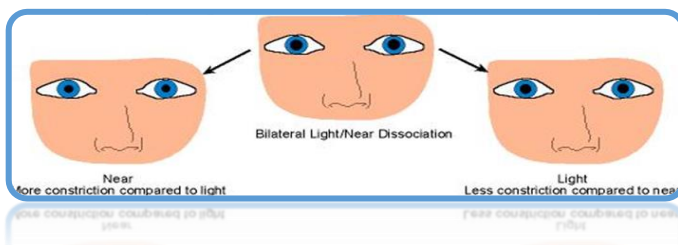
- Caused by denervation of the postganglionic supply to the sphincter pupillae and ciliary muscle follow by viral illness.
- Signs :
 - 1- Unilateral in 80%
 - 2- Affected pupil is large and regular
 - 3- Constriction to near is very slow
 - 4- Redilatation is also very slow
 - 5- Diminished deep tendon reflex

Abnormal Pupillary Reaction

- 1) Afferent pupillary conduction defect
- 2) Horner's syndrome
- 3) **Argyll Robertson Pupil**
- 4) Adie pupil

Argyll Robertson pupils

- Caused by Neurosyphilis
- Signs :
 1. Bilateral and Asymmetrical
 2. The pupils are small and irregular
 3. The light reflex is absent but near reflex is normal (light – near dissociation)
 4. Pupils difficult to dilate



Adie's tonic pupil:

Tonically dilated pupil reacts much significantly to accommodation more than light.

Caused by infection to ciliary ganglion.



Affection of Extra-Ocular Muscles

OCULAR MOTOR NERVES

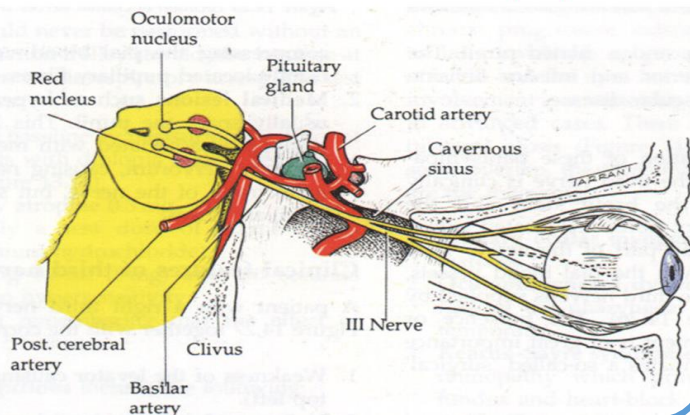
1. Third Nerve
2. Fourth Nerve
3. Sixth Nerve

OCULAR MOTOR NERVE

3rd CRANIAL NERVE

- Oculomotor nerve
- Entirely motor in function
- Supplies –
 - All the Extraocular muscles except superior oblique and lateral rectus
 - Levator palpebrae superioris
 - Intra ocular muscles- Sphincter pupillae and ciliary muscle

Oculomotor Nerve (III)



Oculomotor nerve palsy

Clinical feature

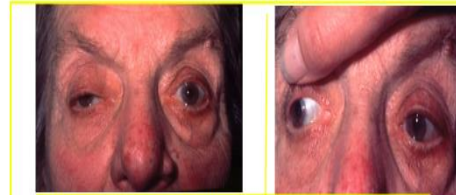
- ✓ Ptosis
- ✓ Abduction of globe
- ✓ Intortion of the globe which increases on attempted down gaze
- ✓ Limitation of adduction
- ✓ Limitation of elevation
- ✓ Limitation of depression
- ✓ Dilated pupil with defective accommodation

Oculomotor nerve palsy

Clinical feature

1. Ptosis due to weakness of levator muscle
2. Exotropia due to unopposed action of LR
3. Intorsion and down due action of SO
4. Normal abduction
5. Limiting elevation due to weakness of SR
6. Parasympathetic palsy causing a dilated pupil

Signs Of Right Third Nerve Palsy



- Ptosis, mydriasis and cycloplegia
- Abduction in primary position

- Normal abduction
- Intorsion on attempted downgaze



- Limited adduction

- Limited elevation

- Limited depression



Clinical Features of IIIrd Nerve Palsy

Ⓢ Symptoms :

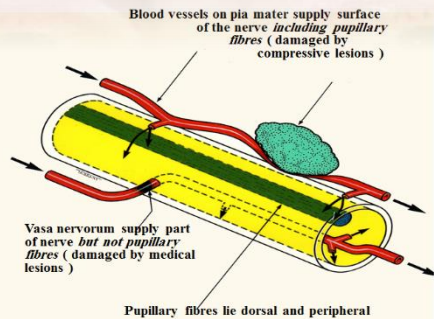
- 1) Diplopia from misalignment of the visual axes
- 2) Symptomatic Glare in bright light (if the ptotic lid does not cover the pupil)

Ⓢ Findings:

- 1) Involved eye usually is deviated down and out (infraducted, abducted)
- 2) Ptosis (differentiated from myasthenia gravis by TENSILLON TEST)
- 3) Pupillary Dilatation
- 4) Paralysis of Accommodation causes blurred vision for near objects



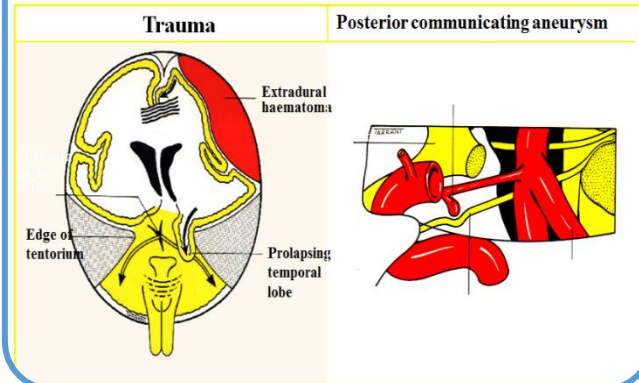
Applied anatomy of pupillomotor nerve fibres



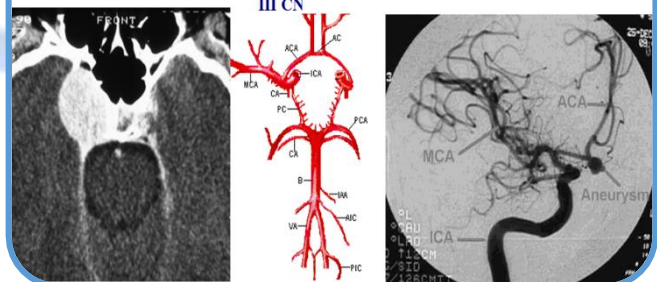
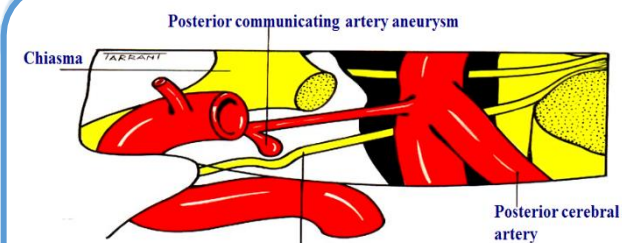
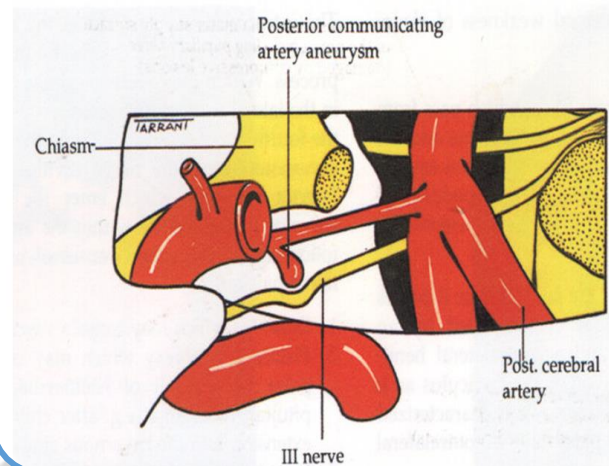
Important causes of isolated third nerve palsy

Idiopathic - about 25%

Vascular disease - hypertension, diabetes



Oculomotor nerve palsy

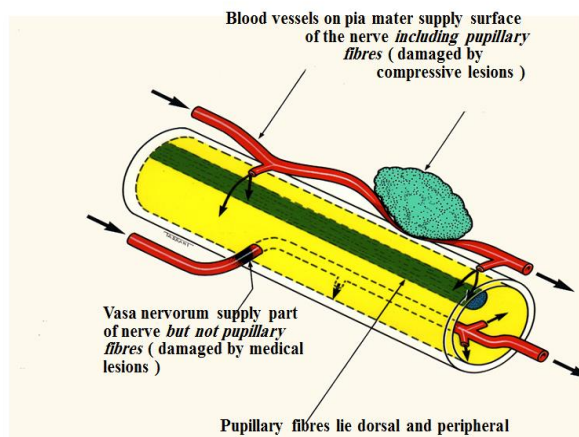


Oculomotor Nerve Palsy

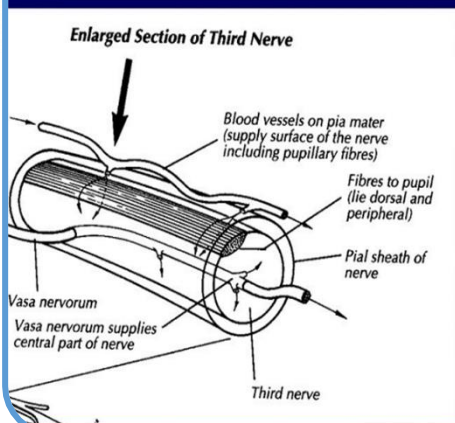
Causes:

1. Vascular disease
 - a. Hypertension
 - b. Diabetes Mellitus
2. Trauma
3. Aneurysm at the junction of the posterior communicating artery with the internal carotid artery (giving isolated painful third nerve palsy)
4. Idiopathic in about 25%

Applied anatomy of pupillomotor nerve fibres



Is the pupil involved?



“diabetic” 3rd nerve – pupil is spared

“surgical” 3rd nerve – pupil is involved

Pupil-Sparing Third Nerve Palsy

- Diabetes, hypertension, hyperlipidemia, smoking, high hematocrit.
- Pupils is spared.
- Pupil involvement reported only in 14%-32% , but anisocoria (difference in pupil size) is **less than 1 mm** (relative-sparing).
- Improve within 4-12 weeks (defer neuro-imaging).

Treatment

Non-surgical

- ✓ Treatment of underlying cause
- ✓ Diplopia – Occlusion patch or prism in involved eye
- ✓ Monitor children for development of amblyopia

Treatment

Surgical

- ✓ Neurosurgery – Aneurysm or haematoma
- ✓ Strabismus or ptosis surgery – Not earlier than 6 months from time of onset

Trochlear Nerve (IV)

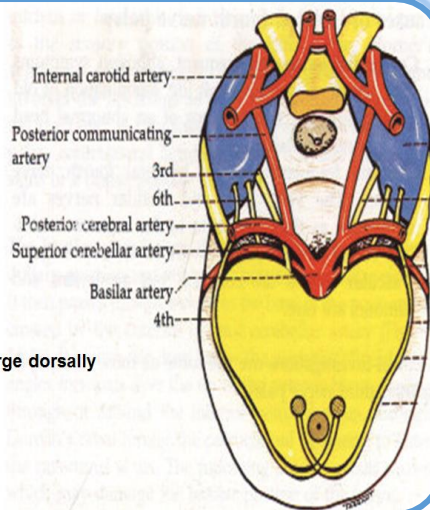
- Important features :
 1. It is the only cranial nerve to emerge from the dorsal aspect of the brain
 2. It is a crossed cranial nerve
 3. It is a very long nerve

Trochlear Nerve (IV)

- The **trochlear nerve [IV]** is a cranial nerve that carries GSE fibers to innervate the superior oblique muscle, an extraocular muscle in the orbit.
- It **arises in the midbrain** and is the **only cranial nerve to exit from the posterior surface of the brainstem**.
- After curving around the midbrain, it enters the inferior surface of the free edge of the tentorium cerebelli,
- continues in an anterior direction in the **lateral wall of the cavernous sinus**, and enters the orbit, through the **superior orbital fissure**.

Trochlear Nerve

- Only cranial nerve to emerge dorsally
- Crossed cranial nerve
- Very long and slender



Trochlear nerve Palsy

- **Clinical features**
 1. Diplopia
 2. Abnormal head posture
 3. Positive Bielschowsky test
 4. Excyclotorsion

Signs of right fourth nerve palsy



- Right hyperdeviation in primary position when left eye fixating
- Excyclotorsion



- Right underaction on depression in adduction
- Vertical diplopia



- Right overaction on left gaze

Fourth Nerve Palsy

- Head tilt (in congenital palsy , it can be overlooked).

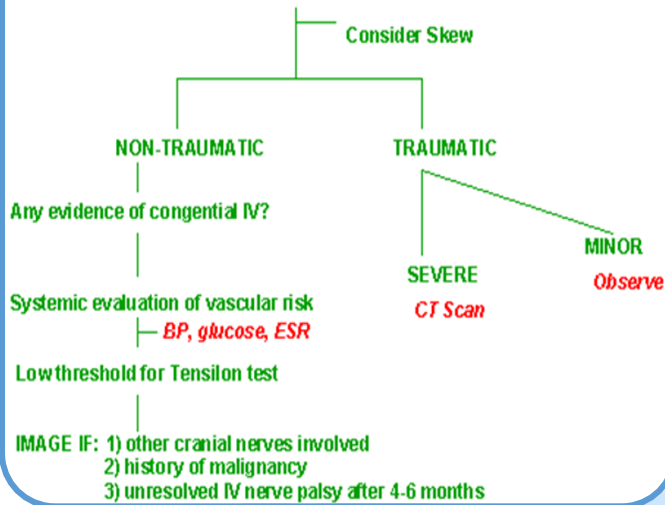


► Diplopia

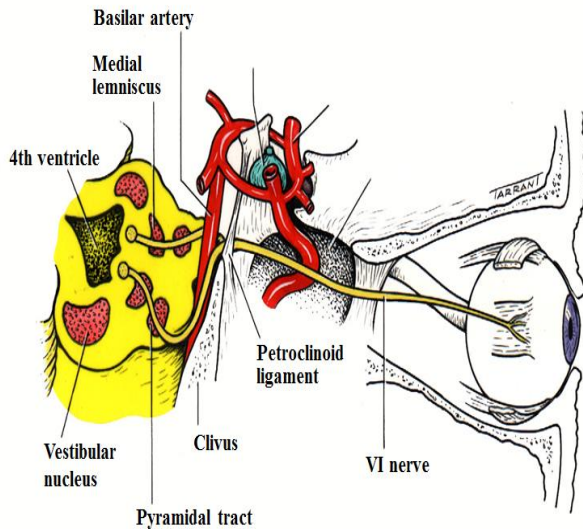
- Patients with a IV nerve palsy typically experience vertical diplopia and in some cases may be aware of cyclotorsion.



Approach to IV Nerve Palsy

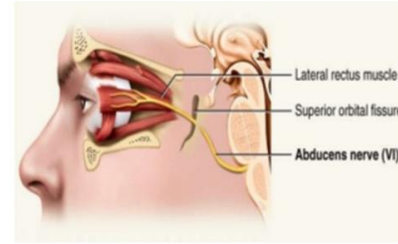


Anatomy of sixth nerve



Abducent (sixth cranial) nerve

- Entirely motor nerve, supplies to lateral rectus muscle.
- Most vulnerable cranial nerve, to damage in traumas involving cranium.

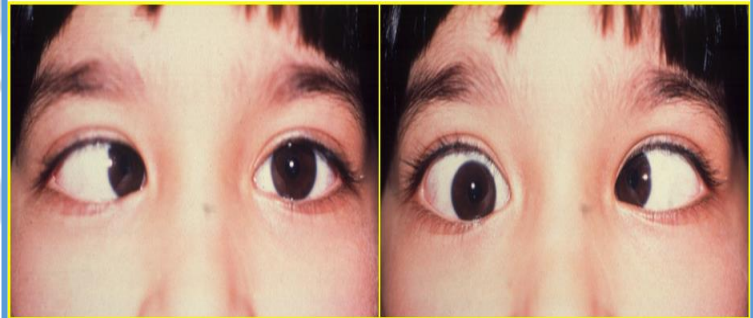


Abducent nerve Palsy

Clinical features

1. **Deviation-** In primary position, eyeball is converged due to unopposed action of medial rectus muscle.
2. **Ocular movements-** Abduction is limited due to weakness of the lateral rectus muscle.
3. **Diplopia-** Uncrossed diplopia occurs, which becomes worse toward the action of paralysed muscle.
4. **Head Posture-** The face is turned towards the action of paralysed muscle to minimise diplopia.

Recent right sixth nerve palsy



Right esotropia in primary position due to unopposed action of right medial rectus

Marked limitation of right abduction due to right lateral rectus weakness

Trochlear Nerve Palsy

Causes

1. Congenital
2. Trauma
3. Vascular (Hypertension and Diabetes)

Left sixth nerve palsy

- convergent strabismus
- failure of abduction on the affected eye (on looking to the paralysed side)
- horizontal diplopia



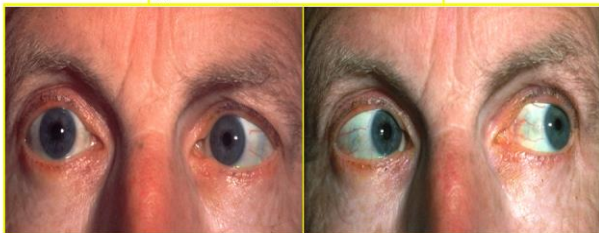
Left sixth nerve palsy
(looking to the left)



Old right sixth nerve palsy



Straight in primary position due to partial recovery



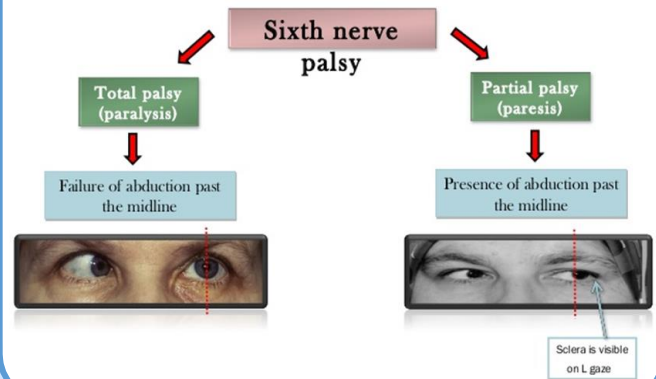
Limitation of right abduction and horizontal diplopia

Normal right adduction

Abducens Nerve Palsy



Type of Sixth Nerve Palsy



Clinical signs & symptoms

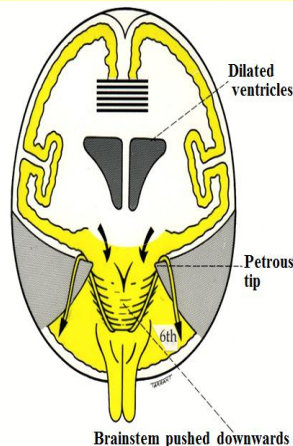
Esotropia	• D>N • Increase with gaze at affected side	
Limitation to abduction	• In the affected eye • Normal adduction	
Horizontal binocular diplopia	• Worse at affected side & distance	
Head-turn	• Towards affected side • To avoid diplopia	

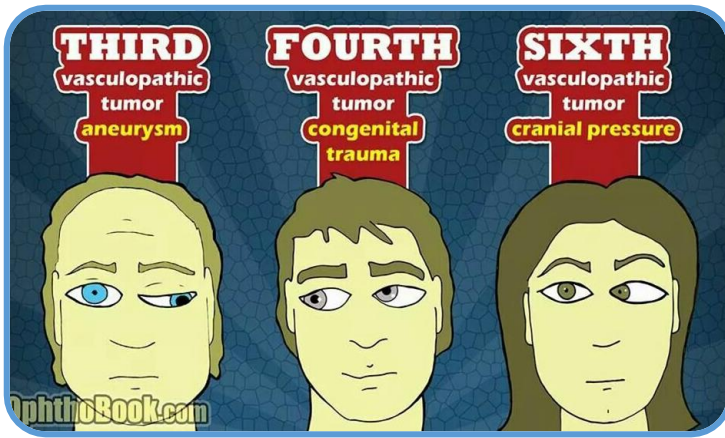
Important causes of isolated sixth nerve palsy

Vascular - hypertension, diabetes

Raised intracranial pressure

Acoustic neuroma





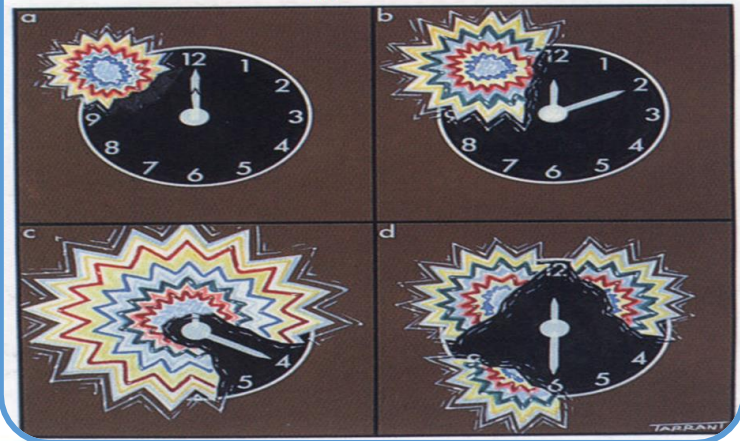
Migraine

- It is often a familial disorder characterized by recurrent attacks of headache.
- Types
 1. Common Migraine
 2. Classical Migraine
 3. Retinal Migraine
- Common Migraine
- Characterized by autonomic nervous system dysfunction
- Premonitory features: change in mood, frequent yawning and poor concentration
- Headache starts as pounding or throbbing then spread to involve half of or the whole head
- Headache lasts for hours to a day or more associated with photophobic and phonophobic.

Classical Migraine

- It is less common but better recognized
 1. The attack is heralded by a visual aura last for 20 minutes characterized by :
 - Bright or dark spots zig-zags scintillating scotoma
 - tunnel vision and hemianopia
 2. Small bright positive scotoma developed then fortification spectrum gradually enlarge.

Migraine



Retinal Migraine

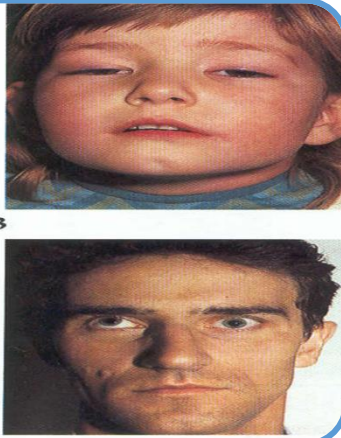
- It is characterized by acute transient loss of vision similar to amaurosis fugax
- Occasionally occur in middle aged patients
- **Management**
 1. Avoid the agents that may precipitate an attack like : coffee, chocolate, alcohol, cheese, oral contraceptive, stress, lack of sleep, long interval without food.
 2. Prophylaxis: beta blockers, low dose of aspirin.
 3. Treatment: NSAID, aspirin, ergotamine

Myasthenia Gravis

- It is autoimmune disorder characterized by reduction of acetylcholine receptors on the end plates of neuromuscular junctions of skeletal muscles .
- Presentation: typically during the third and fourth decades with excessive fatigability of ocular, bulbar and skeletal muscles
- Ocular involvement
 1. Ptosis
 2. Diplopia
 3. Nystagmoid movements

Myasthenia Gravis

Ptosis
Diplopia

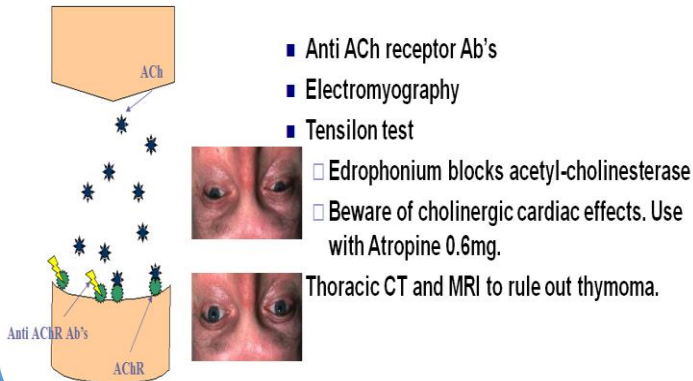


Myasthenia Gravis

- 1) Fatigability
- 2) Double vision
- 3) Lid twitch
- 4) Ptosis
- 5) Normal reflexes & sensation



INVESTIGATIONS



Myasthenia Gravis

• Investigations

1. Edrophonium IV 10 mg
2. Electromyography (EMG)
3. Antibodies to acetylcholine receptors
4. CT or MRI show thymic enlargement in 15%.

Myasthenia Gravis

• Treatment

1. Medical therapy:
 - anticholinesterase (neostigmine)
2. Systemic steroids
3. Azathioprine; reduce anticholinesterase receptor antibody
4. Plasmapheresis
5. Thymectomy

Others

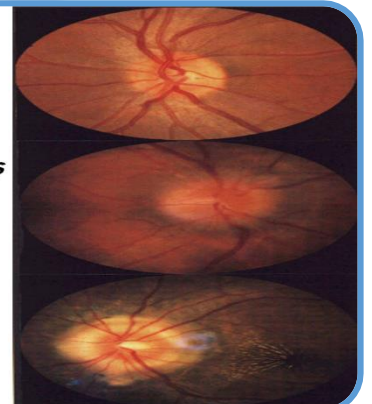
1. Migraine
2. Myasthenia Gravis
3. **Demyelinating Diseases**
4. Increased Intracranial Pressure
5. Intracranial Infections

Demyelinating Diseases

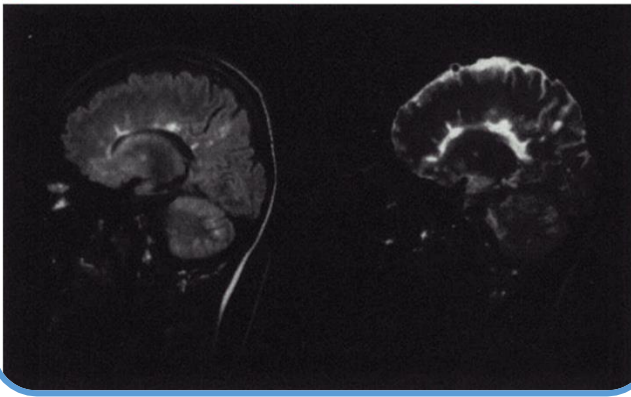
Multiple Sclerosis

1. Optic Neuritis (Retrobulbar)
2. Primary Optic Atrophy
3. Field Defects
4. Paralytic Squint (6th)
5. Nystagmus

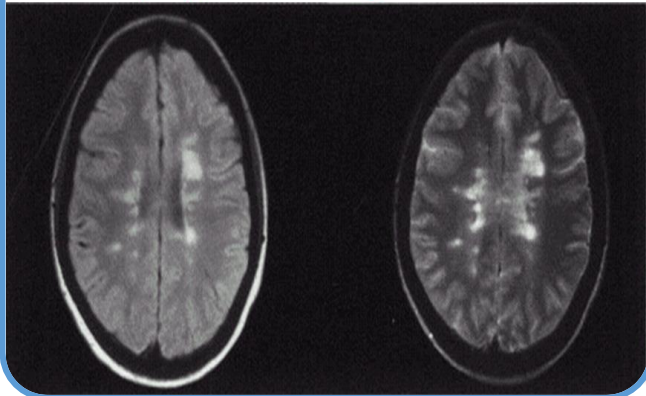
Ophthalmoscopic classification of optic neuritis.
Top: retrobulbar neuritis - normal disc;
bottom: neuroretinitis



Sagittal MRI scan showing periventricular plaques of demyelination.



Axial MRI scan showing periventricular plaques of demyelination



Intracranial Tumours

- General Signs:
 - Headache, Vomiting and Papilledma
- False localizing Sign: 6th CN Palsy
- Endocrine Changes (Pituitary and Pineal body tumours)
- Focal Signs

THANK YOU



Intracranial Infections

1. Meningitis
 - a. Optic Atrophy
 - b. Squint (paralytic or kinetic)
2. Brain Abscess
 - a. Papilledma
 - b. Focal signs
3. Syphilitic Infection
 - a. Papilledma
 - b. Optic atrophy, optic neuritis, field defects
 - c. Argyll Robertson's pupil, Corneal anesthesia

